

MANCHURIAN
PLAGUE PREVENTION SERVICE
MEMORIAL VOLUME

1912-1932

EDITED BY

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Director, Manchurian Plague Prevention Service (1912-1932);

Director, National Quarantine Service

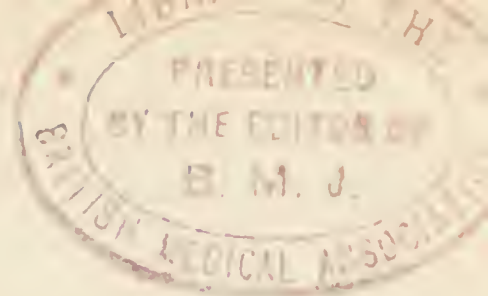
SHANGHAI, CHINA
NATIONAL QUARANTINE SERVICE
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PNEUMONIC PLAGUE

Posterior aspect of Left Lung—natural size—
showing broncho-pneumonic patches

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To the Delegates
attending the
NINTH CONGRESS
of the
FAR EASTERN ASSOCIATION OF TROPICAL MEDICINE
at
Nanking, October, 1934,
this Volume
is respectfully
DEDICATED

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By

THE MERCURY PRESS

17 Avenue Edward VII

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P R E F A C E

Events in this part of the world have marched rapidly since the holding of the Eighth Congress of the Far Eastern Association of Tropical Medicine at Bangkok in 1930. As a result of happenings in Manchuria from the time of the Mukden incident on September 18, 1931, the Manchurian Plague Prevention Service, established in 1912 following the 1910-1911 Pneumonic Plague Epidemic, has passed into other hands. For the first time since the birth of the Republic, the continuity of administration and research, admitted on all sides to be a remarkable feature of public service in this country, has been severed.

The two decades of useful work which have closed witnessed the first systematic organisation of public health in this country. It was a pioneer period in the history of modern medicine on the Asiatic continent, fraught with significance for the subsequent elaboration of State Medicine in China. It ushered in an era of constructive effort. Considerable research was made into the problems of pneumonic and bubonic plague and cholera following the pneumonic plague outbreaks of 1911 and 1921, the bubonic plague epidemics of 1928, 1929 and 1930 in the Tungliao district and the cholera invasions of 1919 and 1926. The results of these and other investigations are embodied in seven bulky volumes published biennially by the Service, the last appearing in 1931. Two unique features of this institution were the Plague laboratory and museum containing perhaps the most complete specimens upon pneumonic plague in man and animals in the world, and a rare scientific library comprising some thousands of volumes.

The present volume consists of a collection of the best original articles that have appeared in the seven volumes of Reports of the Manchurian Plague Prevention Service. It was thought, when this country was selected as the *venue* for the Ninth Congress, that the delegates would welcome an authoritative resumé of the activities of twenty years compressed within the convenient limits of a single volume.

Together with its companion volume published by the National Quarantine Service, this book is dedicated to the delegates attending the Ninth Congress of the Far Eastern Association of Tropical Medicine now in session at Nanking, in the belief that a fair estimate may be derived by these distinguished representatives from the Orient of the sort of work that is being attempted in China in certain aspects of medical research and administration.

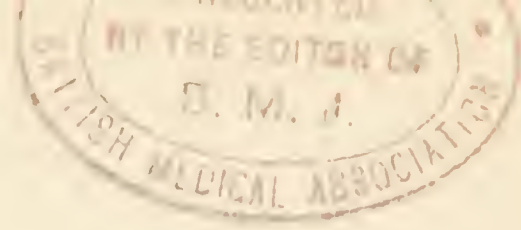
WU LIEN-TEH

Shanghai, October 1, 1934.



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CONTENTS

	Page
Preface	
A Short History of the Manchurian Plague Prevention Service	1
Inaugural Address Delivered at the International Plague Conference Mukden, 1911	13
Notes On the Histology of some of the Lesions Found in Pneumonic Plague	21
The Second Pneumonic Plague Epidemic in Manchuria 1920-21 ..	51
Clinical Observations Upon the Second Manchurian Plague Epidemic 1920-21	79
Pathological Findings in Plague Pneumonia, Second Manchurian Epidemic 1920-21	101
A Study of the Morbid Histology of the 1921 Manchurian Plague Epidemic	125
The Original Home of Plague	143
Summary of Plague Experiments on the Lice of Tarabagans	159
A Systematic Experimental Study of the Pathology of Pneumonic Plague in the Tarabagan and Sisel (Suslik)	163
Plague Transmission Through the Ectoparasites of the Tarabagan	189
Some Remarks Upon the Epidemiology and Histology of Pneumonic Plague	195
New Pneumonic Plague Ward, Harbin, 1926	199
Endoparasites of the Tarabagan	203
The Perpetuation of Plague Among Wild Rodents	207
The Perpetuation of Plague Among Wild Rodents, With Special Reference to the Siberian Marmot	229
The 1919 Cholera Epidemic in China	251
The Cholera Epidemic of 1926	267
Investigations on the Vitality of <i>Vibrio Cholerae</i> on Chinese Paper Money	301
Problems of Cholera in China and Japan	307
Scarlet Fever in China	315
The Scarlet Fever Problem in the Far East	331
The Problem of Venereal Diseases in China	343
Medical Progress in China Since The Republic	363
Early Days of Western Medicine in China	369
How I Built Hospitals in China	397
Abstract of Nosological Articles	409
Blood Grouping in North-Eastern Mongolia and in North Manchuria	421
List of Wild Rodents Known or Suspected to Suffer from Plague	423
Early Chinese Travellers and Their Successors	437
APPENDIX	
Wu Lien-teh—A Short Autobiography	459

A SHORT HISTORY OF THE MANCHURIAN PLAGUE PREVENTION SERVICE

BY WU LIEN-TEH 伍連德

Director

*Manchurian Plague Prevention
and National Quarantine Services*

The terrible epidemic of pneumonic plague which invaded Manchuria and North China in 1910-11, though it exacted a toll of 60,000 lives and caused monetary losses estimated at 100 million dollars, definitely laid the foundation for systematic public health work in China. Those in authority from the Emperor downwards, who had formerly pledged their faith to old-fashioned medicine, now acknowledged that its methods were powerless against such severe outbreaks. They were thus compelled to entrust the work to modern-trained physicians and to give their consent to drastic measures, such as compulsory house-to-house visitation, segregation of contacts in camps or wagons, and cremation of thousands of corpses which had accumulated at Harbin and elsewhere.

Incidentally, it may be recorded here that soon after his arrival at Harbin, Dr. Wu performed on December 28, 1910 *post-mortem* on the corpse of a female Japanese inn-keeper at Fuchiatien (native city) and established the presence of pneumonic plague lesions besides obtaining pure cultures of *B. pestis* therefrom.

For the first time in Chinese history, an International Medical Conference was called together at Mukden in April, 1911, under the chairmanship of Dr. Wu Lien-teh, the deliberations of which shed much light upon the almost unknown problem of pneumonic plague. It was attended by such well-known scientists as Richard Strong, S. Kitasato, D. Zabolotny, A. Stanley, Paul B. Haffkine, L. Padlevsky, G. Shibayama, A. Fujinami, Oscar Teague, C. Broquet, Reginald Farrar, Erich Martini, Gino Galeotti, O. Gonzalez-Fabela, S. T. Zlatogoroff, G. Koulecha, Dugald Christie, J. Chabaneix, etc.

Its deliberations were embodied in a fine "Report of the International Plague Conference, 1911," covering 500 pages and published by the Bureau of Printing, Manila.

The most gratifying result, however, was the establishment of the North Manchurian Plague Prevention Service as recom-

mended by the Mukden Conference. The main resolutions passed by the Conference in regard to the creation of such a Service were as follows:—

13. The need for isolation of pneumonic plague patients being urgent, permanent isolation hospitals should be available. Such isolation hospitals should admit of individual isolation, be of rat-proof construction, and be capable of easy disinfection.
42. A permanent sanitary nucleus should be formed, capable of rapid expansion in time of plague, and a list should be drawn up of medical officers who could be sent immediately to the affected area on the outbreak of plague.
44. With the view of giving effect to these recommendations, every effort should be made to organize a central public health department, more especially with regard to the management and notification of future outbreaks of infectious diseases.

The establishment in October 1912 of the Manchurian Plague Prevention Service, with headquarters at Harbin, was a serious attempt made by the Chinese Government to give effect to the recommendations of the Mukden Conference mentioned above. Its inauguration was somewhat delayed by the Revolution, which had started in October of the previous year, but fortunately the Viceroy of Manchuria (Chao Erh-sun), the Inspector-General of Customs (Sir Francis Aglen), the Vice-Minister of Foreign Affairs (Dr. W. W. Yen), and the Commissioner of Customs (Mr. W. Haines Watson), all took a keen interest in the matter and did what they could to promote its success. For instance, the Viceroy appropriated from the Manchurian revenue ¥50,000 for the hospital at Harbin, ¥40,000 for Manchouli, ¥30,000 for Tsitsihar, and ¥20,000 for Lahasusu.

Sir Francis Aglen induced the Diplomatic Body of Peking, which at first vetoed the scheme, to change their minds and to agree to the withdrawal of ¥60,000 annually from the Chinese Maritime Customs for the maintenance of the Service; Dr. W. W. Yen showed his sympathy by drawing up some of the regulations and placing the Plague Prevention Service under the aegis of the Waichiaopu (Foreign Office); lastly Mr. W. Haines Watson (Commissioner of Customs, Harbin, died 1914), who was present throughout the great plague of 1910-11 and therefore fully understood the urgent need of preventive measures, gave invaluable advice in the organization of the several hospitals at the beginning of their existence.

A large piece of land (120 mow) lying between the Railway Area and native city in Harbin was presented by the Governor of Kirin. The Harbin Plague Prevention Service Hospital, construction of which began in September 1911, was completed in summer 1912. The original plant, erected at a cost of \$70,000 and opened in December 1912, consisted of two separate com-

pounds, the west containing buildings devoted to administration and the treatment of general patients as well as the quarantining of four hundred persons; the east compound containing isolation blocks for the accommodation of thirty suspects and forty plague cases. A modern administration block, which temporarily housed also the laboratory and the operating theatre was opened in 1919. In 1922 another new block was added for the accommodation and treatment of general patients, followed in 1924 by an up-to-date building to house the laboratory, library and museum. In 1926 a model pneumonic plague ward was erected in the East compound permitting of the accurate observation and treatment of patients with a maximum of safety to the staff.

Simultaneous with the erection of the Harbin Hospital, plans were made for the construction of a similar institution in the boundary town of Manchouli which had been the gate of entry of the 1910-11 epidemic. In 1911 the Viceroy of Manchouli (Chao Erh-sun 趙爾巽) gave £40,000 for building a hospital at Manchouli. Foundations were forthwith dug in the spring of 1912, and much building material was purchased, but during the succeeding Revolution the Mongols, fanned by Russian expansionists, burnt our place and left nothing behind but the bare ground. Several years then passed, and it was not until the second Manchurian Plague epidemic of 1921 came that we were able to lease some buildings from the Municipality for our preventive work. Since that time a permanent sanitary staff has been stationed at Manchouli. In 1923 we bought a ready-built stone house for \$9,000 for the purpose of a research laboratory and quarters for our medical officer, at the same time retaining a large wooden block lent by the Municipality as hospital. At this station was also installed the large apparatus for disinfecting tarabagan skins with formalin gas before export.

In the same year as the original Harbin hospital (1912) an isolation hospital was opened at Lahasusu (同江) at the junction of the Sungari and Amur Rivers, followed in 1913 by a similar establishment at Sansing (三姓) on the Sungari River, and in 1914 by a large plant at Taheiho (大黑河) on the Amur River opposite Blagovestchensk.

The scope of the work was increased when in 1918 a special appropriation was sanctioned for the establishment of a Quarantine Hospital at Newchwang. Building operations were commenced in 1919 and the hospital was formally opened on July 10, 1920. There is a large front block one hundred and sixty-two feet wide, containing operation and diagnostic rooms and general wards; next is a disinfection block; and behind lies the contagious block, with individual rooms and verandas facing

the south. The cost of these original buildings was ¥40,000, accommodating forty-five beds. In 1923-24, a series of six detention blocks, built of brick and having cement floors, were added to the hospital. Each possesses a set of hygienic k'angs (sleeping platforms), insect-proof and dirt-proof, to serve as beds for those detained under observation. There is accommodation for eighty persons in each block; hence, a minimum of four-hundred persons may be detained at any one time. The cost of his second lot of buildings was \$40,000. In the year 1927 \$9,000 were spent to add a new block for medical examination of passengers.

North Manchuria being up to the year 1919 free from major epidemics, the energy of the Service was devoted during the first years of its existence to general medical work as well as to an investigation of plague problems in Manchuria and Transbaikalia. The results were embodied in the biennial Reports of the Service, the first of which appeared in 1914. Seven bulky volumes have appeared since.

In 1919, when cholera invaded Harbin, the Plague Prevention Hospital concentrated its energy upon the fighting of this epidemic with most gratifying results. Out of 1,962 patients admitted, only 275 (i.e. 14.11%) died, though many came in a practically moribund condition. In 1920-21 the Service was confronted with the task of fighting the second Manchurian pneumonic plague epidemic, this disease having once more entered China from its haunts in Eastern Transbaikalia into the adjacent Manchurian territory. Though, for reasons beyond the control of the medical staff the promising endeavours to localise the outbreak in Hailar were frustrated, and the disease once more reached Harbin, the radical measures taken succeeded in limiting the total number of victims (including Siberia) to 9,300 and in practically staying the spread of the epidemic south of Harbin, which city had played an ominous role in the 1910-11 outbreak.

During the 1920-21 epidemic an exhaustive study of pneumonic plague was made. After this, researches upon the tarabagans (Siberian marmots), suspected to be the reservoir of the disease, were continued with redoubled vigour and led to encouraging results. Joint work of the Plague Prevention Service staff with the Russian Anti-Plague Detachment in Transbaikalia definitely established in 1923 the existence of plague epizootics among the marmots, while independent investigations of the Chinese scientists at Manchouli succeeded in demonstrating experimentally the role of the tarabagan flea in the transmission of the disease (1924). These results as well as an exhaustive compilation of the literature to date were embodied in a voluminous "Treatise on Pneumonic Plague" by

Wu Lien-teh, published by the League of Nations Health Section in 1926.

Through further researches upon tarabagans (published in 1928) it became possible to demonstrate experimentally that plague is carried over the winter in hibernating animals leading to acute disease with bacteremia after their awakening in spring.

Although the plague focus to the west of North Manchuria appeared to have become quiescent since 1928, a new responsibility was added to the tasks of the Service by perennial outbreaks of bubonic plague in the Tungliao and adjacent districts of South Manchuria. A comprehensive study of the situation was therefore made by members of the staff in 1928. The main burden of dealing with the 1929 and 1930 outbreaks in rural communities among a backward population also fell upon our Service. Plague reappeared in 1931, but our field work was cut short by the Japanese invasion of Manchuria. Fortunately the outbreak was limited.

Activities at the various stations were continued though much hampered and at times actually endangered by the hostilities. The 1932 cholera outbreak found us well prepared. As on previous occasions, the presence of the disease at Harbin was first diagnosed in our laboratory and ample use was made of our vaccine. The Municipal (former Russian) Hospital having no experienced bacteriologist on its staff, our experts had to supervise the laboratory work there in addition to carrying out our own.

In spite of these manifold and essential activities the newly established Customs authorities at first refused to continue the modest appropriation which had been regularly received for twenty years, though they afterwards paid out a much smaller sum. The name of the Director, who had been faithfully carrying out the work since its inception, was arbitrarily struck off the list by the new Japanese Commissioner. The activities of the Service, which had hitherto built up an international reputation among scientific circles with its up-to-date research institute and unique plague museum, have been practically strangled. Only simple routine hospital work is now done by a junior officer in charge. It may truly be said that the Manchurian Plague Prevention Service continues to function in name only. Fortunately such an *impasse* had been anticipated ever since the commencement of the Japanese invasion on September 18, 1931, and arrangements had been made to carry on the work in Shanghai under new conditions but with unimpaired *morale*.

A list of major publications by various members of the Service since its establishment in 1912 is herewith appended:

APPENDIX

LIST OF IMPORTANT PUBLICATIONS OF THE
MANCHURIAN PLAGUE PREVENTION SERVICE

Abbreviations used:

- Rep. I=Reports 1911-13 of the North Manchurian Plague Prevention Service, Cambridge, 1914.
 Rep. II=Dto. for 1914-17, Peking, 1917.
 Rep. III=Dto. for 1918-22, Tientsin, 1922.
 Rep. IV=Dto. for 1923-24, Tientsin 1924.
 Rep. V=Dto. for 1925-26, Tientsin, 1926.
 Rep. VI=Dto. for 1927-28, Tientsin, 1928.
 Rep. VII=Manchurian Plague Prevention Service Reports, 1929-30, Tientsin, 1930.
 F.E.A.T.M.=Far Eastern Association of Tropical Medicine.
 Ch. M.Jl. =China Medical Journal.
 N.M.Jl. =National Medical Journal of China.

A. PLAGUE

- | | |
|---|--|
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| — | Investigations into the Relationship of the Tarabagan (Mongolian Marmot) to Plague.
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&
Woodhead,
Reynolds, | Notes on the Histology of some of the Lesions found in Pneumonic Plague.
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| Wu Lien-teh,
& | Transmission of Pneumonic and Septicemic Plague Among Marmots. |
| Eberson, | Rep. II, 7-17. Am. Jl. Inf. Dis., 1917, 20, 170. Jl. Hyg., 1917, 16, 1. |
| Eberson | Plague Poisons and Virulence.
Rep. II, 18-22. Am. Jl. Inf. Dis., 1917, 20, 180. |
| — | On the Nature of Plague Proteotoxins.
Rep. II, 23-28. N.M. Jl. 1917, 3, No. 1, 10. Am. Jl. Inf. Dis., 1917, 21, 56. |
| — | Active Immunity to Systemic Plague Infection.
Rep. II, 29-44. N.M. Jl., 1917, 3, 125. Am. Jl. Inf. Dis., 1918, 22, 62. |
| Wu Lien-teh, | Plague in the Orient with Special Reference to the Manchurian Outbreaks (Address delivered at the Opening of the Union Medical College, Peking, September, 1921). Jl. Hyg., 1922, 21, 62. |
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Rep. III, 1-54, Jl. Hyg., 1932, 21, 262. |
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Pollitzer, | Observations made during and after the Second Manchurian Plague Epidemic of 1920-21.
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Rep. III, 83-98. JI. Hyg., 1923, 21, 329.
- Clinical Observations upon the Second Manchurian Plague Epidemic 1920-21.
Rep. III, 120-141. JI. Hyg., 1923, 21, 289.
- Pathological Findings in Plague Pneumonia, Second Manchurian Epidemic 1920-21.
Rep. III, 142-165.
- J. W. H. Chun, Salient Points about the 1921 Pneumonic Plague in Harbin.
Rep. III, 166-180. N.M. JI., 1922, 8, 71.
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- Yang Ting-kuang & W. H. Shih, Scarlet Fever in China.
Rep. IV, 207-223.
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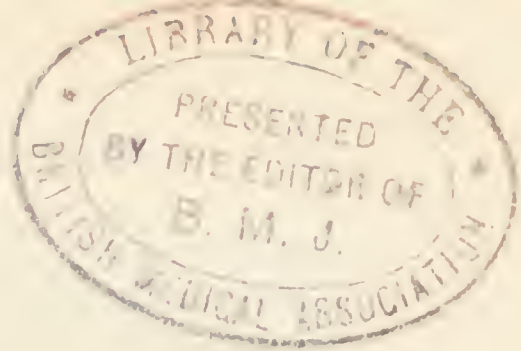
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INAUGURAL ADDRESS DELIVERED AT THE INTERNATIONAL PLAGUE CONFERENCE, MUKDEN, 1911

BY WU LIEN-TEH

Director

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Permit me to express the great pleasure it gives me in accepting the appointment as Chairman of this International Plague Conference, consisting as it does of so many well-known names in the medical and scientific world. I feel very diffident in having to address such an august body, well knowing my poor qualifications, and the only reason I can think of for having this honour thrust upon me is the success of the plague preventive work in Harbin, in which, however, I seem to have but a small share, in comparison with the arduous tasks which fell to my staff of able and willing assistants.

As His Excellency the Imperial Commissioner has remarked, this pneumonic plague sprang upon us suddenly, and, despite all our efforts, has claimed up to this date more than 46,000 victims. It seems that this form of plague is not actually new to Manchuria, as for many years, certainly within the last decade, there have been sporadic outbreaks both among Russians and Chinese in Siberia, Mongolia and Manchuria. There are statements that it raged during the seventeenth century, but so far as can be gathered from really authentic records this is the first extensive outbreak of the exclusively pneumonic variety that has occurred for ages. You may be aware that the epidemic which swept over Newchwang in 1899 was mainly bubonic in character, as also was that which killed over 800 people in Tonghan in 1908. We are indebted to the Russian doctors for the first accurate information regarding epidemics of pneumonic plague, and, as full papers will be read before this Conference by our learned Russian colleagues, I will pass over that part of the subject and confine myself rather to what little we have been able to discover regarding the present outbreak. That a certain rodent, the *Arctomys bobac*, known in English as the marmot, in Russian as the tarabagan, and in Chinese as the *han ta*, is chiefly associated with this form of plague, has for some time been fully believed by the inhabitants of Mongolia and north-western Manchuria. According to some valuable information

gathered by Dr. Ch'uan, of the Chinese Medical Staff, who was sent up to the station of Manchouli to report on the subject, it appears that the local people have long been familiar with this disease, both in men and in animals. From the actual marmot hunters themselves he found out some items which, I venture to believe, will exercise an important bearing on our knowledge of the plague.

Nature is very rich in coincidences, and perhaps as scientists more than any other class of men you are prepared for such, but who could have dreamed that the healthy marmot, basking, as it loves to do, in the warm sunshine, utters a cry resembling the sound of "*Pu p'a, Pu p'a*," which in the Chinese language, at any rate, means "don't be afraid," or "no harm." Sickness renders it mute, so that in the light of present knowledge it would seem that when the marmot is not crying "no harm, no harm," there is very real harm indeed. The sickness in the tarabagan, which we presume is the forerunner of the plague, in this case is characterized by an unsteady gait, inability to run or to cry when chased, and, when caught, the physical signs are seen to consist principally of enlargement of the glands. When noticing the above signs the experienced hunter leaves his quarry severely alone and betakes himself to more distant spheres. During the past few years, however, there has been an exceptional demand for the marmot skins in the European and American markets, and numbers of wandering Shantung coolies have, in consequence, found their way to the marmot resorts, hunting the animal indiscriminately, and food being scarce, they have often cooked and eaten the flesh of the marmot. A number of these Shantung men have died in previous years, but not in sufficient numbers to attract attention. The season for killing marmots is from August to the middle of October. About the third week in October of last year there were said to be fully 10,000 of these hunters gathered in Manchouli and Hailar with their skins, waiting to sell them and then to return south for the winter. Dr. Ch'uan found out that the symptoms of sickness suffered by these hunters were headache, fever, and spitting of blood-coloured sputum, followed by rapid death.

The tarabagan, or marmot, has a wide range over the plains and sandy wastes of Mongolia and Central Asia, and now that such attention has been called to its power of infecting human being with plague, we shall learn more about its habits. We know that it hibernates and that in spring the younger ones seek new homes in which to breed. The new burrows often run into old "earths," in which it may happen that there may be dead left from a previous season which may infect the new arrivals. The animals that die in the open are probably all devoured by birds, etc., but it seems that the marmot creeps into its home,

as a rule, to die. The inexperienced hunters nearly always dig out the marmots from their holes and thus run more risks than the ordinary Mongol, who generally hunts the marmot in the open, or traps it near its abode, thus coming into contact with and catching only healthy animals. Despite the apparent risk, however, there are no statistics which show that many hunters die on the plains, but when they are gathered together in the late autumn at the market places they crowd into very poor hovels or inns, where, with piles of raw pelts, there may often be found from twenty to forty in number, sleeping and eating, in the smallest of most badly ventilated rooms, wherein the conditions are ideal for the encouragement of an epidemic.

From Chinese sources we learn that the first case of pneumonic plague reported in Manchuria occurred in the small town of Manchouli on October 12, 1910, but owing to the energetic action of the Russian authorities only about 400 died; the last case reported was on December 25—i.e., two and a half months after the outbreak. The first cases reported in Harbin were on November 7, when two tarabagan hunters, who had arrived from Manchouli, were lodged in the shop of an artesian well mechanic. These two men died of plague, and infected four others with whom they lived. From this time forth the epidemic began to spread rapidly, and Harbin soon earned an unenviable reputation as a plague-distributing center, from whence infection spread into central and southern Manchuria, and far into the northern provinces of Chihli and Shantung.

The following few dates are of interest as showing the rate at which certain centers became infected:

Place	Date.	Place.	Date.	Place.	Date.
Manchouli	Oct. 12, 1910	Hsinminfu	Jan. 14, 1911	Kirin City e	Jan. 16, 1911
Harbin	Dec. 4, 1910	Yungpingfu b	Jan. 15, 1911	Chingchowfu	Jan. 14, 1911
Shuangchengpu	Jan. 5, 1911	Chefoo c	Jan. 21, 1911	Tientsin	Jan. 15, 1911
Kuanchengtze	Dec. 14, 1910	Tsitsihar	Dec. 4, 1910	Peking	Jan. 12, 1911
Mukden	Jan. 2, 1911	Hulanho d	Dec. 13, 1910	Tsinanfu	Feb. 1-7, 1911

a. Number of cases considerably increased.—(Editor.)

b. Not on railway.

c. By sea from Dalny.

d. Fifteen miles from railway.

e. Eighty miles from railway.

For the accuracy of the dates I can not vouch, but they are correct within a day or two. It is instructive to see—

(1) That the plague tended to follow the quickest line of travel.

(2) That the lines of infection corresponded exactly with the routes taken by the coolies on their return home for the Chinese New Year.

(3) That although Shuangchengpu is only about 30 miles south of Harbin, on the Russian Railway, the first case reported

there was nearly two months after the outbreak in Harbin. When it did appear it was extraordinarily virulent, killing over 4000 people in the whole district in the course of two months.

(4) That it spread to certain towns—e.g., Newchwang, also Chinwangtao, the principal winter port of north China. The reason in the former case appeared to be due to the fact that the harbor is frozen in winter; in the latter case the cause might be traced to the coolies not using it on their southward march by foot. That Tongshan actually, and Peking and Tientsin practically, escaped might be due to the employing only of local coolies in winter, and, therefore, those homeward bound from Manchuria would not remain in these cities.

As the main object of this Conference is expressed in the words of His Excellency the Commissioner, "We are determined hence-forth to meet this enemy, the plague, armed with the best knowledge we can obtain," I will, therefore, venture to lay before you the following topics for your consideration: I would request you to confine your attention, as far as possible, to the present epidemic in its pneumonic, septicaemic, and other forms (if such be existent), and only to deal with the bubonic form, in so far as it throws light upon the present outbreak, and I trust that the elucidation of some of the many plague problems at this Conference will enable us more effectually to deal with it in the future.

It would be idle on my part, at this stage, to attempt in any way to deal *ad seriatim* with the many interesting problems—some of them exceedingly obscure—which have arisen out of this epidemic, but a few observations made while I was in Harbin may perhaps be of interest to you. When I first went there many of the inhabitants with whom I conversed informed me that this epidemic was quite an ordinary visitation, to which they had been accustomed. It never killed many, and would stop of itself, if no attention were paid to it. From both Russian and Chinese records there is no doubt that the pneumonic form of plague had occurred there before, but why on this particular occasion it should have assumed such a virulent form, killing, within the space of three months, over 5000 persons in a population of 30,000, I hope you may be able to explain.

Two factors seem to have contributed largely to the virulence of the epidemic in the Chinese city. These were, first, the severe climatic conditions, the thermometer registering, at times, —30° C., which extreme cold prevented people going out of doors, and, secondly, the low, dark, dirty and overcrowded houses, which formed the majority of the dwellings. At the same time it is worthy of note that some of the double-storied houses, with plenty of air space and not overcrowded, were also badly infected.

In one house, particularly, a porcelain shop, situated in the largest street, containing eight inmates including the proprietor, none of whom belonged to the coolie class, one after the other had the plague, until in the end not a person was left to claim the property.

While on this topic I should like to say a few words on the character of the epidemic at Shuangchengpu, a city situated on the railway line, 30 miles south of Harbin. As cities are known in China, there could be no greater contrast than between Shuangchengpu and *Fuchiatien* (the Chinese town of Harbin). The latter is closely packed and built on a low-lying, swampy plain, with narrow streets, inhabited principally by coolies, while the former is a finely planned city with wide streets at right angles to each other, some of which are as much as from eighty to a hundred feet broad. This town is famed for its spacious compounds and large, well-constructed houses. There is much open space used by the numerous large inns, bean mills, distilleries, pawnshops, etc., and at least half of the population is well to do. There is little poverty among the people. The majority have settled and live in families, thus making a very marked contrast to the transitory hand-to-mouth multitude that forms so large a section of *Fuchiatien*. About half of the families of Shuangchengpu are Manchus, many of whom are wealthy landowners who, with the leading Chinese merchants, are very clean in their habits and homes. Yet there were 1500 deaths in this city of about 60,000 inhabitants within the space of seven weeks. I cite this to show that here may be other reasons as well as dirt and poverty to account for the fierce mortality.

Turning to the subject of statistical data, full tables will in time be handed to you for your consideration. It is interesting to note that where records have been kept the age of greatest susceptibility is from 20 to 40 years. In *Fuchiatien* less than 100 women died out of a total of over 5,000 deaths, while in Shuangchengpu nearly 500 out of 1,500 deaths were women. This is explained by the fact that in *Fuchiatien* there are almost no females, while in Shuangchengpu a large part of the population is made up of families.

This epidemic found us at the beginning relying on many of the data of bubonic plague relating to prevention and treatment, but the results of our experience at *Fuchiatien* have tended to modify our early expectations as to the efficacy of the vaccines and serums procured by us. Another fact perhaps worthy of notice is that in Mukden alone 13,000 rats have been examined, besides large numbers in other centers, under the Chinese administration, without showing any signs of plague. It might be suggested, therefore, that few, if any, have been infected; yet in Harbin the unusually large number of deaths among domestic animals, as also the deaths of about 400 horses and

300 pigs, might suggest the possibility of a similar epidemic infection.

I regret deeply that the enormous amount of work in hand, at the time when the epidemic was raging, and our limited number of assistants made it impossible for us to collect positive scientific facts upon this subject.

The question of natural immunity forced itself upon our consideration. Several plague-hospital assistants and attendants worked right through the epidemic in hospitals where scores were dying daily under our notice, even though the precautions taken by them were of the lightest character and were even non-existent over long periods of time.

Two items of interest which proved their utility during the Harbin outbreak in such a manner as to have an important bearing upon any future epidemic may be alluded to: First, the use of railways wagons for quarantine work; and, second, the disposal of the dead bodies by burning.

The ease with which a railway wagon, holding at the most not more than twenty persons, enables a segregation camp to be divided into small units completely isolated from each other, also the simplicity of disinfection, the early detection of suspects, the satisfactory ventilation by small windows and sliding doors, and the heating by a central stove, suggest at once a most efficient form of quarantine which can be quickly established at any place in proximity to a railway.

The burning of the dead, which, though not unheard of in Chinese annals, still conjures up in the native mind all that is repulsive and contrary to natural feelings, yet once decided upon by the Government, was accepted by the people without complaint or hindrance.

At a time when severe frost made burial impossible, burning, by its ease and simplicity, commended itself to all of us. A pit, 20 feet square and 10 feet deep, which had been blasted by dynamite, was capable of holding 500 bodies at a time. When bodies were in coffins the wood of the coffins was sufficient for complete burning, but bodies without coffins required four pieces of wood 2 feet long by about 4 inches in diameter for each body, and upon the whole mass in the pit, kerosene oil was pumped from a fire engine at the rate of ten gallons for every hundred bodies. This, when lighted, burnt so rapidly and fiercely that little or nothing remained but ashes. The example of *Fuchiatien* in this respect, was afterwards followed by neighbouring towns, and the process has proved to be a satisfactory means for the disposal of dead bodies in all large epidemics.

Such an action on the part of the Chinese Government in dealing with the great problems of the plague, and its permission

to conduct *post-mortem* examinations upon unclaimed bodies for the further investigation of the disease, granted for the first time in the history of China, must prove to you that the Government is moved by the highest motives of humanity, and is ready to lay aside age-long prejudices, to spend money unsparingly, and to possess itself of all that science can impart for the saving of life and the elimination of national perils.

In closing, may I remind you that this is the first International Medical Conference held in China, and it is impossible to estimate its widespread effects. Besides the beneficial results of your observations and resolutions on the subject of plague, the impulses you will set in motion, by the fact of this Conference, will react not only upon the national life but more particularly upon the future progress of medical science in China, and I feel deeply the burden of the honour placed upon me in being Chairman of this medical conference, which is unique in our history, powerful in its representation, and which gives China a strong position among nations seeking the welfare of their people.

NOTES ON THE HISTOLOGY OF SOME OF THE LESIONS FOUND IN PNEUMONIC PLAGUE

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AND

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The lesions of bubonic plague and of plague produced experimentally have been fairly fully described, especially with regard to the naked-eye appearances of the lymphatic glands, lungs, liver, kidney, spleen, and heart; but it was not until comparatively recently, in the outbreak of the great pneumonic plague in Manchuria, that an opportunity occurred of studying at all fully the histology of the lesions met with in this condition. Since this outbreak, however, a number of observers, working in the plague field, or on material supplied by these workers, have dealt somewhat fully with the pathological anatomy and histology of primary and secondary pneumonic plague, and have made no slight contribution to the hitherto scanty literature of the subject.

Our material is from patients who succumbed to pneumonic plague in the Manchurian epidemic. At first, the lesions of the heart and liver attracted our attention as most requiring investigation; but later we determined to examine the whole of the material at command and we now describe it in the following order:—lung, bronchial glands, heart muscle, liver, spleen, and kidney.

We make no attempt to generalise as to the types of lesions met with in pneumonic plague patients, but offer a note of the actual conditions seen in material prepared in Manchuria and examined at Cambridge, and of those naked-eye appearances only that are presented in small pieces of tissue transmitted to Cambridge. We then compare our findings with those of certain other observers.

The material available for examination consisted of a fragment of heart muscle from a case of pneumonic plague;

a fragment of liver from a case possibly, but not necessarily, the same as the above; and pieces of well-preserved lung, bronchial glands, spleen, liver and kidney tissue from a second (or a third) case. It is impossible to say much concerning the naked-eye appearance of the solid organs, of which only small pieces, fixed and hardened in formalin and formol glycerine, are available, but a few points of interest are noted, as each organ is dealt with.

We think it well, however, before dealing with our own material, to give a short account of the appearances described by different investigators, especially as considerable differences of opinion exist amongst them as to the exact nature of the lesions—particularly of the lesions found in the lung—in cases of pneumonic plague.

LUNG.—Wilm (1897²³) examined the lungs from a number of his Hongkong cases in 1896. Intense congestion, oedema, and well-marked infiltration were found in the lower lobe in five cases, evidently pneumonic. The bronchi were usually unaltered though in some there was congestion of the mucous membrane with a considerable quantity of mucus on the surface.

Wyssokowitz and Zabolotny (1897²⁴) speak of the pneumonia as of a broncho-pneumonic type accompanied by bronchitis, the foci in the primary plague pneumonia becoming numerous and confluent in the later stages, producing a lobar consolidation. They describe a secondary pneumonia resulting apparently from the extension of the disease from the blood-vessels. These observers never found the pneumonia occupying the whole lobe of a lung. In the small and medium-sized bronchi the mucous membrane was red and covered with greyish mucous fluid sometimes stained with blood and mixed with air.

Yamagiwa (1897²⁵) describes the catarrhal pneumonic condition in the consolidated patches of which the vessels are congested. The interalveolar septa are infiltrated with red blood corpuscles and leucocytes, similar cells filling many of the alveoli, in which filaments of fibrin and degenerating alveolar epithelium may also be seen. Almost pure cultures of plague bacilli may be found in the blood-vessels and in the alveoli.

Albrecht and Ghon (1898-1900¹) also describe primary plague pneumonia as a typical lobular or broncho-pneumonia, usually most marked in the posterior portion of the lung, the patches of consolidation sometimes becoming confluent though, frequently, areas of congested but air-retaining lung lie between them. These authors draw attention to the fact that the pleura over the consolidated areas may be slightly cloudy or deeply injected, that there may be numerous small haemorrhages or—in this the condition differing essentially from an ordinary catarrhal pneumonia—the pleura may be covered with a layer of fibrinous lymph. They describe a very characteristic appearance of the septa of the alveoli which become much broadened, appearing as a glistening homogeneous framework which stains well with eosin. In this network are a few cells or cell nuclei and red blood corpuscles. They also note the disappearance of the interalveolar septa at certain points and that the dilated bronchi contain blood-stained fluid in which enormous masses of bacilli are embedded, but they point out that fibrinous exudate is almost absent, only a few solitary fibrils or a very delicate network being seen at wide intervals.

Childe (1898⁵) draws attention to the intense congestion and oedematous condition of the lungs; to the congestion of the bronchial mucous membrane and to the frothy watery fluid, sometimes blood-stained, that can be squeezed from the bronchi. He describes a number of pneumonic patches varying in size from that of a pea to that of an egg, light pink, red grey, or deep blood red in colour, solid and airless, rounded in shape and separated from the crepitant lung by a distinct ring of engorgement. Those of the patches which were situated on the surface of the lung projected from the surface whilst "the pleura over them was roughened and showed signs of early inflammation." He likens such patches to those of the 1st and 2nd stages of ordinary lobular pneumonia. There never was any softening or breaking down. Sometimes these pneumonic patches were much larger and occupied a considerable part of the lobe. Petechial haemorrhages were sometimes present on the surface and the "bronchial glands were either enlarged, swollen, oedematous, soft and distinctly engorged" or else about the normal size and only slightly engorged. In a section of the lung the large blood-vessels and the interalveolar capillaries, between the pneumonic areas, are greatly engorged; there are also small haemorrhages into the alveoli between which the engorged vessels run.

He describes the pneumonic areas very much in terms of a broncho-pneumonia, dividing each area into three zones. In the outer of these there is intense engorgement of all the blood-vessels and capillaries in the walls of the alveoli, many of these alveoli being filled with blood. In some cases the alveolar septa are broken down and may be represented by mere shreds of tissue. In an intermediate zone the alveoli are intact and are filled with well-stained cells, evidently catarrhal, whilst in the centre of each pneumonic patch the alveoli are so stuffed with these cells that it is difficult to distinguish the outlines of the alveolar walls. In any or all of these zones haemorrhages may be present. The intermediate zone usually contains, in addition to the catarrhal epithelium, a few leucocytes and red blood corpuscles with, now and again, a few filaments of fibrin. In the dense central area catarrhal cells and leucocytes with some granular debris fill the alveoli. There is great congestion of the walls of the bronchial tubes and engorgement of the large veins in the walls of which haemorrhages are seen; and he states that "blood and catarrhal cells may be seen in the finer bronchi but the bronchial mucous membrane is scarcely altered, there being at most a little cellular proliferation. There are the appearances of acute pleurisy over those pneumonic areas which project upon the surface of the lung with haemorrhages beneath the pleura."

In the report of the Indian Plague Commission (1901²⁶) the following interesting passage occurs: the lesions in primary pneumonic plague, "when contrasted with those occurring in *Pestis major*,....elsewhere than in the lungs are less intense, while those in the lungs are more intense" in which the large as well as the small blood-vessels are intensely congested and "haemorrhagic zones are seen scattered throughout the lung, filling the alveoli and often breaking down their walls. Within the haemorrhagic zones are areas in which the alveoli are completely filled with leucocytes, epithelial cells, and granular debris constituting, with surrounding zones of haemorrhage, blood congested areas of catarrhal pneumonia." The Commissioners point out that the bronchi are engorged with blood and that catarrhal cells are found at the terminations of these passages, and also that over the affected areas at the surface of the lung the pleura may be acutely inflamed. Agreeing with Major Evans (1901⁸), Captain Elphick (1901⁷) and Major Jones (1901²⁶), they consider that croupous or lobar pneumonia is the form that occurs most frequently. The pneumonia is described as occurring in "small detached patches constituting lobular areas only when the

inflammation has not far advanced; but it is lobar to the extent of involving a whole lobe or the greater part of a lobe when the lung inflammation has advanced further." Or again several cases are described "in which individual lobes or even an entire lung was consolidated." The Commissioners also lay stress on the fact that in many cases only slight changes were found in the bronchi and they suggest "that the pneumonia is lobular in patients who have died at an early stage of the disease and lobar in those who have survived to a later period; or, otherwise, that lobar pneumonia occurs when the toxin is most virulent and most widely distributed throughout the lung, and lobular pneumonia when it is less virulent and less widely diffused."

Hassan Hamdi (1904¹³), describing primary plague pneumonia, follows Yamagiwa pretty closely but points out that the alveolar epithelium even when desquamated contains in its substance red and white corpuscles and plague bacilli, whilst vacuoles can be seen in their protoplasm. Only a few isolated strands of fibrin can be seen in the alveolar contents. Bacilli may be found in the interalveolar septa and in the alveolar cavity. Where the bacilli are numerous the alveolar epithelium appears to be disintegrated, the mononuclear and polynuclear cells usually being collected in the centre of the alveoli where they are often surrounded by a ring or wreath of bacilli. The nuclei of the leucocytes are broken down, the interalveolar and interlobular septa are thickened, homogeneous and contain few nuclei, though they may be infiltrated with bacilli and with small round cells. Later this infiltration becomes so marked that the vessels can scarcely be seen and even the connective tissue fibrils are obscured. The most advanced changes and the earliest may be seen almost side by side in the same lung. The larger vessels of the lung are distended and congested, the tunica adventitia at first oedematous, later becomes infiltrated with leucocytes and bacilli. No special changes appear in the tunica media but in the connective tissue layer of the tunica intima there is a round cell infiltration. In many of these vessels the endothelium is detached or much swollen. Bacilli are comparatively few within the vessels; in the capillaries a number of giant cells similar to those met with in the bone marrow are sometimes found. The larger lymphatics contain red and white blood corpuscles, but little fibrin; the endothelial lining cells may be swollen. The connective tissue of the pleura, especially in the deeper layer, appears to be thickened and homogeneous, later it may become infiltrated with cells and there may be a laying down of fibrin and a wandering of leucocytes from the distended vessels. The pleural layer of endothelial cells has sometimes disappeared. In the later stages of the pneumonia the lymphatics in the pleura may be filled with bacilli. This author describes secondary pneumonia as resulting from an extension of the disease from the blood-vessels to the septa and the alveoli. In these cases the blood-vessels are distended and filled with blood and the bacilli are very numerous, far more so than in the primary cases. In the alveolar septa the number of leucocytes is comparatively small but there are often many mast cells. In these cases, too, the alveolar epithelium is usually desquamated and the number of bacilli in the vessels may be so great that they form a kind of colourless clot; then the walls of the vessel giving way the bacilli make their way into the alveoli. The oedematous alveolar septa and alveoli contain numerous red blood corpuscles and bacilli and resemble those in a haemorrhagic infarcted lung. They sometimes contain an albuminous fluid which, on coagulation, is homogeneous and transparent. In this fluid many bacilli lying in groups or scattered singly between the cellular elements are found. Numerous alveolar epithelial cells, swollen and multinucleated, may be found alongside leucocytes. This desquamation and swelling of the alveolar cells is so characteristic a feature in the secondary pneumonias that where it is marked it may be

accepted as being almost pathognomonic. The desquamated vacuolated alveolar epithelium shows in its protoplasm ingested bacilli, leucocytes and red blood corpuscles, and pigmented granules.

Strong (1912²⁰), describing material taken from twenty-five autopsies made within a few hours after death, notes that delicate fibrinous adhesions are often observed between the parietal and visceral pleurae; also punctiform haemorrhages sometimes becoming confluent and forming larger dark red haemorrhagic patches. Fresh fibrinous pleurisy is present in every case, sometimes as a delicate reddish membrane. In other cases the fibrin is "greyish, or greyish-white and could be easily pulled from the surface of the lung; rarely, a gelatinous, oedematous, exudate was present. In two instances the pleural cavity contained between 100 and 200 c.c. of blood-stained fluid in which large numbers of plague bacilli were present." There are numerous ecchymoses beneath the pleura, and pneumonic infiltration and engorgement of some portion of the lung, the pneumonic areas being surrounded by zones of marked congestion and oedema from which a large quantity of reddish, serous fluid can be expressed. The pneumonic areas are either lobular or lobar in type, circular in outline or wedge-shaped and are usually paler at the centre than at the periphery. The mucous membrane of the bronchi is deeply congested. The broncho-pneumonic areas contain no air; cut sections have a dry, harsh and sometimes granular appearance and no mucous plugs can be expressed from the bronchi. An "entire lobe in a state of grey hepatisation such as is frequently found in ordinary croupous pneumonia due to the *Diplococcus pneumoniae*" is never met with, probably because the patient dies before this stage is reached. In one lung patches of lobular consolidation are noted while in another a whole lobe is involved. In the bronchi is a red, frothy, bloody, serous fluid, or a reddish mucous exudate, it is always blood-stained and often contains plague bacilli. In a later paper Strong in conjunction with Crowell and Teague (1912²¹) gives a somewhat fuller account of the histology of this condition. In no case examined were the lungs free from pathological change, bacteria occurring in enormous numbers especially about the bronchioles in the earliest cases, in the peribronchial lymph spaces and in the adjoining alveoli. They frequently form masses completely encircling the bronchiole and are present in large numbers in the interlobular septa beneath the pleura, following very closely the distribution of the carbon pigment present in such large quantities in these lungs. In the cases that succumb quickly few bacilli are found in the blood and although the alveoli contain bacilli the lining epithelium is, as yet, but slightly altered. The blood-vessels and capillaries are widely distended, are sometimes ruptured and smaller haemorrhages may be seen around them. There is catarrhal inflammation of the smaller bronchi and bronchioles, swelling and some desquamation of the epithelium and a few red blood corpuscles and leucocytes may be found amongst the epithelium and in the lumen of the bronchioles; on the surface, mucus and large numbers of bacilli may be found. In this early stage the alveoli may be almost filled with bacilli and contain a few desquamated epithelial cells, serum, and an occasional leucocyte or red blood cell. In the later stages a larger number of leucocytes and red blood corpuscles are found in the alveoli, the red corpuscles always preponderating, though the leucocytes, both polymorphonuclear and mononuclear, become more numerous as the disease advances. Few, if any, eosinophile cells are met with. There is fragmentation of the nuclei of the leucocytes. The leucocytes, when seen under high magnification, are very frequently, surrounded by a clear zone. Phagocytosis was seldom observed. Strong comments upon the absence of fibrin from the exudate in most cases and maintains that, when present, it is found in small quantities only. In the later stages of the disease the bacilli are very numerous especially in the medium-sized vessels, in the lymphatics

of the deep layer of the pleura and in the fibrinous exudate which always covers the pleura over the consolidated areas.

The mucous membrane of the bronchi is intensely congested and thrown into a series of longitudinal folds.

Dr. Akira Fujinami (1912¹¹) made 29 autopsies in Manchuria, three of them on animals. He considers that intense congestion and great oedema of the lungs characterised the pneumonia and preceded a form of hepatisation which is peculiar to plague. The lung is harder to cut but not very compact, the surface of a section is not so granular as in ordinary croupous pneumonia and the consolidation is very patchy, sometimes dark red, sometimes greyish-red or greyish-yellowish-red; the red tinge is seldom absent. In some cases there are no well-developed areas of local hepatisation; the lungs simply look congested. Any part of the lung may be affected but the consolidation was noted most often in the right upper lobe, then in the left upper lobe and then in the lower lobe. In only one case was the right middle lobe the special seat of the hepatisation. On histological examination hyperaemia of the blood-vessels and capillaries of the lung is observed; serous fluid, numerous leucocytes and red blood corpuscles and desquamated epithelial cells in variable quantity are present in the alveoli of the hepatised area. Fibrin threads within the alveoli are rare or almost absent and in the non-hepatised areas an oedematous fluid containing a few leucocytes may fill the alveoli. Plague bacilli are numerous in the alveoli, not only in the hepatised areas but where there is no inflammatory cellular exudation. Bacilli also occur in and around the blood-vessels and bronchial walls, in the perivascular and peribronchial lymphatics and in the sub-pleural tissue. Single bacilli or small colonies are seen in the blood-vessels whilst other organisms sometimes accompany the plague bacilli. This author, with Strong, describes a fibrinous membrane covering the pleura especially over the hepatised areas.

Dr. Koulecha (1912¹⁴) describes 28 cases and speaks of a true fibrinous lobar or pleuro-pneumonia, the pleura always being covered with a fine fibrinous layer, although, as he admits, there is a lack of fibrin in the exudate into the alveoli, this exudate consisting of a serous fluid in which a variable number of red blood corpuscles and leucocytes appear, the proportion of these different corpuscles determining to some extent the depth of the colour of the pneumonic patch. The desquamation of the epithelium of the alveoli is not a marked feature and the exudate is not catarrhal in character. This exudate contains an enormous number of plague bacilli which also accumulate both in the perivascular lymphatics and in the blood-vessels whence, with their products, they pass from the blood-vessels into the alveoli of the lung.

Signorelli (1913¹⁹) set out to ascertain whether in plague the pneumonia is fibrinous or of the catarrhal type. He quotes Lustig and Galeotti (1897-1901¹⁶) and Galeotti (1900¹²) as able to produce intravascular coagulation by means of injections of the endotoxin or protein of *Bacillus pestis*. The so-called nucleoproteids of plague injected into animals in suitable doses, they say, reproduce functional and histological changes similar to those that follow an ordinary infection, Federici (1898⁹), injecting these substances through the wall of the thorax of the rat or the rabbit into the substance of the lung, found that they set up great congestion of the interalveolar capillaries and damage and slight proliferation of the endothelium lining the alveoli, but he describes no increase of fibrin. Galeotti, injecting frogs, with the same material, into the lung substance as above, and guinea-pigs by the trachea, observed numerous haemorrhages and fibrinous clots in the alveoli. Examining the lung from a case of primary pneumonic plague sent from Mukden, Signorelli found, however, that in the *dilated* alveoli cells of

various types were embedded in a network formed of delicate threads of fibrin. In the alveoli the epithelium is markedly disintegrated though a few swollen cells with feebly staining nuclei adhere to the alveolar wall, and he insists that the alveolar content is only partly fibrinous, that the meshes in the network of fibrin are large and that the cells occupying them are crowded together and of various types:—lymphocytes, large mononuclear cells and polymorphonuclear leucocytes, the latter few in number, many of the large cells having a feebly staining protoplasm and a swollen vacuolated-looking nucleus. In the polymorphonuclear cells the nucleus is often undergoing pyknotic changes. Many of the large mononuclear cells are loaded with blood pigment; the exfoliated and degenerated epithelial cells of the alveolus may often be seen fairly distinctly. The bacilli are scattered in the alveoli between the cells of the exudate, rarely within them, phagocytosis, as far as the bacilli are concerned, being ill marked. Here and there are well-defined groups of bacilli lying free between the cells and around them a few fibrin filaments may be seen. One of the chief points noticed is the rapid desquamation of the alveolar epithelium, sometimes before it has time to proliferate. An important feature in all these preparations examined by Signorelli is rupture of the thrombosed and fragile capillaries and consequent breaking down of the alveolar septa. In some cases the bacilli appear to be multiplying not only in the alveoli but also in the lymphatics of the lung tissue as well as in the blood-vessels. Signorelli carried out a series of experiments on dogs, injecting the lungs with plague toxin or the nucleo-proteid of the plague bacillus, and showed that it produced localised areas of inflammation, diffuse hyperaemia around these areas and, usually, well-marked oedema of the pulmonary tissue; the vacuolated and degenerating epithelium covering the alveoli in such artificial injections was often desquamated, although no living plague bacilli were present. He looks upon the lesions in pneumonic plague as the result of an inflammatory process with fibrinous exudate and localisation of the fibrin in the pulmonary capillaries, this being due to the toxic and coagulative action of the nucleo-proteid of plague bacilli, and he lays it down that the thrombosis of the interalveolar capillaries is one of the factors which render pneumonic plague so serious. Sudden diminution of the respiratory area is produced by the blocking of the alveoli and of the capillaries and this is accompanied by a rapid and intense intoxication of the heart muscle, the toxin produced in the lungs passing rapidly, directly, and in large quantities into the chambers of the heart and, acting on the muscle, adds to the circulatory difficulties produced by the blocking of the pulmonary capillaries.

It is evident from these descriptions that in plague we have to deal with at least two types of pneumonia, and from a careful study of the literature and of the material now under examination we are satisfied that there are modifications even of these types. This we will consider after we have given a description of our material in which the following are the appearances presented.

LUNG. There is some slight opacity of the pleura but on the surface of none of the pieces at our disposal is there even a trace of fibrinous lymph. At the pleural extremities of the trabeculae are deeply pigmented patches in which, however, there is little fibrous tissue formation such as is usually associated with the presence of carbon pigment. In most of the blocks there are no haemorrhages or petechiae either on the pleural surface or in the lung substance, but in one small piece three petechial haemorrhages about $\frac{3}{4}$ " apart may be seen at the margin of the lung, probably a piece of the anterior border. Here and there are

distinctly consolidated patches, some of the size of a millet seed, some as large as a filbert, the latter predominating. Even from these areas a little air may be squeezed along with the preserving fluid, though the lung appears to be distinctly collapsed. As the pieces of lung are comparatively small, seldom more than 2" in length and 1" in breadth, it is impossible to determine the relative distribution of these patches. In one piece, apparently from the thin border near the base of the lung, the tissue has quite the normal, spongy, lung consistence and large quantities of air are, on pressure, squeezed out along with the fluid. Some of the large branches of the pulmonary artery seen on the cut surface contain distinct coagula or thrombi; in the consolidated areas this is well marked. A segment of the wall of a bronchus, about $\frac{1}{4}$ " in diameter, is seen in one of the blocks. The mucous membrane, considerably congested, is thrown into folds and is covered with a thin layer of slightly blood-stained mucus.

HISTOLOGICAL EXAMINATION OF THE LUNG. At no point is there any evidence of a fibrinous pleurisy—there is no trace of fibrin on the surface—but a layer of well-preserved endothelial cells is seen either detached from the subjacent fibrous tissue or remaining *in situ* on the surface of the pleura.¹ In these cells the chromatin network of the nucleus and a single or a double nucleolus are well seen; the protoplasm is vacuolated. The fibro-elastic tissue of the pleura is slightly thickened. The lymphatics and lymph spaces of the deep layer of the pleura are greatly distended with fluid or with dark brown or black granular pigment; the blood-vessels are greatly congested. Beneath the pleura and near the surface of the lung are alternate areas of collapse and emphysema. In the interalveolar septa of the collapsed areas there is intense congestion of the capillaries and considerable thickening of the basement membrane on which the alveolar epithelium rests; there is also some slight proliferation of the cells lying in the collapsed air vesicles; some of these proliferating cells contain brown pigment. Around the blood-vessels, large and small, in the lymphatics and in the deep layers of the pleura, in the peribronchial lymphatics and in the lymphatics of the fibrous trabeculae brown and black granular pigment may be seen in considerable quantities. In the depth of the lung the patches of collapsed alveoli are even more distinctly marked than immediately under the pleura. Here the catarrhal pigmented cells are very numerous, and the vessels are greatly congested. From the congested bloodvessels red blood corpuscles have escaped into the collapsed alveolar spaces. The swollen basement membrane, the fenestration of which is evident, of the walls of the alveoli looks as though it had absorbed a considerable amount of fluid. In areas where the collapse is not marked the air vesicles are distended with a coagulated albuminous fluid usually al-

1. This is evidence of the excellent state of preservation of the tissues, which must have been placed in the fixing solution almost immediately after the death of the patient.

most homogeneous, sometimes filled with little vacuoles, or again but rarely, containing a few delicate threads of fibrin. Leucocytes, accompanied or not by a few red blood corpuscles, may be seen lying free in these alveolar spaces, but there is no characteristic fibrinous lymph coagulum with white and red corpuscles such as is usually met with in the red hepatisation of the lung in lobar pneumonia. The process is rather one of intense congestion, collapse, and slight proliferation of epithelium and degeneration with marked oedema. Away from the pleura and from the collapsed patches large numbers of catarrhal cells, many of them containing carbon pigment, are seen in the alveolar spaces. In few of the pneumonic patches can traces of any granular fibrin be demonstrated by Weigert's fibrin stain. Lying in the delicate granular network, when present, there may be a number of mononucleated epithelial cells which appear to be derived by a catarrhal proliferative process from the epithelium lining the alveoli. In a few of the small collections of fibrin there is commencing mucoid change. Throughout our sections of the lung there is evidence of an oedematous condition—vacuolation of the cells, swelling and separation of fibres, and opening up of lymph spaces.

The consolidation of the lung tissue is very patchy, but where the alveoli are not collapsed or filled with proliferated epithelial cells they contain a hyaline albuminous fluid—evidence of an oedematous condition. In the alveoli in which this is most marked the plague bacilli are also most numerous. In the capillaries between the alveoli the walls are distinctly thickened and hyaline, there is often evidence of great activity of the nuclei, marked thickening of the bodies of the endothelial cells and, along with this, thickening of the delicate collagenous tissue—basement membrane—on which these cells lie. Many of the capillaries are ruptured. Dark brown and black pigment of the same character as that described as occurring in the bronchial glands is present in the lymphatics of the interlobular septa, whilst a number of small, fibroid, nodules with deeply pigmented areas in which are few connective tissue or other cells are scattered throughout the section and are evidently the result of chronic irritation set up by inhaled carbon and dust particles. This though a chronic condition is apparently progressive. There is distinct pigmentation of the large and other fibrous septa, and in the perivascular and peribronchial tissue.

The appearances presented in the large bronchi are interesting. Here and there the lining epithelium has almost disappeared, apparently by a process of rapid desquamation, from the basement membrane which is swollen, homogeneous

and hyaline looking. Elsewhere, in place of desquamation, there is marked proliferation of the epithelial cells similar to that described by Hamilton as occurring in acute bronchitis and giving rise to the presence of numerous oval, "peg-top shaped" and rounded cells. The mucosa of the bronchus is thrown into folds through contraction of the peribronchial muscle. In some of the bronchi the columnar epithelial cells are distinctly more "goblet" in character than usual, the nucleus being pushed to one side, large droplets of mucus filling up the body of the cell. Sometimes the droplets of mucus have not run together but form a kind of foamy mass within the cytoplasm. On the basement membrane on which the columnar cells appear to rest may be seen flattened cells not forming a continuous layer but occurring at intervals; in these cells the nuclei are more solid than are those of the other epithelial cells which are distinctly vesicular with a network of chromatin at the margin. In some of the larger branches of the bronchi smooth or slightly granular albuminous material may fill up the whole lumen. Beneath such a clot the epithelium is almost intact but seems to be throwing off large quantities of mucus from the large goblet cells. Debove's layer is also well seen, the basement membrane beneath being much swollen. There is slight cellular accumulation at certain points beneath the basement membrane. The pigment contained in the large cells and in the lymphatic spaces in the wall of the bronchus has evidently entered by way of the alveoli.

The thickened basement membrane in the acutely inflamed larger bronchi is very well brought out in the haematein and van Gieson stained specimens. The columnar and pear-shaped cells usually associated with an acute bronchitis are also specially well defined, though the superficial layer of cells has, in some instances, been completely removed. The thickened basement membrane seems to form an almost impenetrable layer though the distended vessels beneath come very close to its under surface. The vessels in the submucosa are greatly engorged. In some of the smaller bronchi the epithelium is well formed and almost intact, large globules of mucin which have absorbed fluid distending some of the cells.

The peribronchial lymph spaces contain much albuminous fluid. This same fluid has given rise to great thickening of the walls of the alveoli in the immediate neighbourhood of the bronchus. Around the smaller bronchi are localised accumulations of lymphocytes and polymorphonuclear leucocytes evidently called up chemiotactically.

In the bronchi the *Bacillus pestis* sometimes forms a regular layer on the surface of the epithelium, almost pure

cultures being seen in the mucus covering them. They are found usually near the surface but, here and there, they appear to penetrate between the cells and down to the swollen basement membrane, which, however, forms a distinct barrier between the surface and the deeper connective tissue of the wall of the bronchus, and we cannot convince ourselves that there is a single bacillus in the substance of this basement membrane though in the vessels that come quite close to its under-surface plague bacilli are easily demonstrated. They are also seen attached to, and even inside the endothelial cells lining the arterioles in the walls of the bronchus. Except in the large bronchi where pure cultures of the bacillus may sometimes be seen a *comparatively* small number of bacilli are met with in the respiratory area, though, as already noted, small groups may be seen in the alveoli. At certain points, however, near the oedematous areas the disintegration of the cells and their nuclei is very marked, some of these cells being invaded by large numbers of bacilli, the breaking down cell being replaced by a mass of them. Bacilli are also seen in the perialveolar lymph spaces where they have evidently been carried by cells some of which also contain pigment. Here and there, what appears under the low power to be hyaline albumen, when examined under a higher magnification, is seen to consist of an almost pure culture of bacilli filling an alveolar space. Even in the collapsed alveolar spaces the plague bacilli are fairly numerous, most of them lying free, a few of them adherent to the large hyaline cells, but comparatively few being taken into the substance of the cells. In the albuminous (oedematous) coagula however the large phagocytic cells with more numerous contained bacilli may be seen.

In the larger branches of the pulmonary artery are well-formed blood thrombi embedded in which are a number of hyaline mononuclear cells. In the muscle fibre of the walls of these vessels swelling and vacuolation is very evident, whilst the endothelium lining the vessels is in a condition of cloudy swelling and is distinctly vacuolated. These endothelial cells usually stand out very distinctly. Bacilli are so numerous in some of the large vessels that masses of them looking like fibrinous clots may be seen under the low power. In the thrombotic clot bacilli may be seen, sometimes along the margins, in others embedded in it. In a vessel where the former arrangement is evident the bacilli are attached to the endothelium and are attacking it, and may even have brought about its complete disintegration, leaving a roughened fibrillar surface into which the bacilli may be seen making their way between the bundles of connective tissue and muscle fibre. Bacilli are seen in the *vasa vasorum*. They are certain-

ly more numerous in the blood-vessels than in any other position except perhaps in the bronchial secretion and in a few alveoli, especially those containing coagulated albumen and cells. To the clots where the bacilli are numerous many leucocytes have been attracted, but where the bacilli are few the clot is almost devoid of leucocytes. In some of the medium sized and smaller vessels and in the interalveolar capillaries, the bacilli in the ante mortem clots may be so numerous that they can be demonstrated in the haematein and van Gieson stained preparations. The endothelium in the blood-vessels is often in an advanced stage of cloudy swelling and may be the seat of a peculiar vacuolation, this, in some instances, causing a great increase in their size. Pyknosis of the nucleus is often noted. Bacilli may also be seen in the congested vessels of the deep layer of the pleura and a few in the perivascular and other lymphatics in this position. It is noteworthy that wherever the plague bacilli are seen lying on or near a basement membrane this membrane is almost invariably swollen and markedly hyaline. In the pigmented fibrous tissue bacilli may be seen in capillaries and also, though much more rarely, in the lymph spaces between the fibrous bundles.

In the congested interalveolar capillaries, the outlines of red corpuscles can rarely be made out, the capillaries standing out as yellow homogeneous irregular lines from which small haemorrhages are taking place into the surrounding alveoli. The interalveolar septa are considerably thickened, this partly from swelling of the basement membrane of the alveolar wall and partly from thickening of the basement membrane of the walls of the capillary vessels. The epithelial cells lining the alveoli are usually undergoing some proliferation leading, along with the collapse, to partial consolidation of small lobular areas. In these proliferating or catarrhal cells and in the epithelium lining the collapsed alveoli the nuclei are distinctly vesicular and the cytoplasm is vacuolated, but beyond this there may be no evidence of degeneration.

Hyaline cells with rounded and kidney-shaped nuclei may be seen in the alveolar spaces; some of these large cells contain ingested granules of black and brown pigment. In the alveoli and alveolar passages near the bronchi, in which there is an oedematous effusion, there is almost invariably a similar homogeneous exudate.

In the blood-vessels the phagocytic cells, especially the mononuclear cells, have taken up large numbers of plague bacilli but the alveolar epithelium in the collapsed and catarrhal areas seems to have a certain slight power of ingesting even the *Bacillus pestis*, and in some of the cells, one or two bacilli

have become enveloped in the cell protoplasm. Where the bacilli are numerous the nuclei both of leucocytes and epithelial cells take on a lighter stain, sometimes becoming distinctly vesicular or, again, undergoing pyknotic changes.

BRONCHIAL LYMPHATIC GLAND. The macroscopic changes usually described in the bronchial glands of patients suffering from pneumonic plague are somewhat indefinite.

Strong, Crowell and Teague^{20, 21} lay stress on the point that the bronchial glands near the bifurcation of the trachea show more advanced changes than do those in any other position. They maintain that they are "always swollen, rich in blood and frequently almost black in colour from the results of haemorrhages." Fujinami¹¹ confirms this and Koulecha¹⁴ maintains that genuine buboes enormously rich in bacilli are found in these glands, indeed he maintains that they are very like the glands met with in other positions in bubonic plague. He notes that the bronchial glands always contain enormous numbers of plague bacilli, are softened, greatly congested, and embedded in oedematous tissue.

Childe⁵ describes a similar condition in these glands, but he also notes, and in this he is corroborated by Yamagiwa²⁵ and Aoyama³, that these glands are not intensely affected whilst in some there may be no marked changes of any kind.

The bronchial gland of the lung here examined is, undoubtedly, slightly enlarged and congested; moreover it is deeply pigmented, but to the naked eye there is no evidence of haemorrhage or of any of those acute changes described by Childe and by Strong, Crowell and Teague.

On microscopic examination the appearances presented are fairly characteristic. Immediately under the capsule in the cortical sinuses a comparatively small number of plague bacilli may be seen, some lying free in the open network, others applied to the surface of the flattened endothelial cells lying on the swollen strands of the adenoid reticulum. In the vessels of the gland both large and small, as was to be expected, bacilli may be seen either lying free in the lumen, embedded in clot or in contact with the endothelial lining. It is difficult to say that they are contained within the endothelial cells, they look rather as though they are adherent to a viscid surface. Indeed, even the bacilli associated with the deeply pigmented cells appear in many cases to be adherent to their surface rather than to be taken into their substance. These bacilli are somewhat obscured by the presence of large quantities of dark brown or black pigment which has also been taken up by the endothelial cells lying on the swollen trabeculae of the adenoid reticulum. The swollen trabeculae look as though they have absorbed a considerable amount of fluid. Although there is fairly well-marked congestion of the vascular tissue of the gland there are no haemorrhages and there is certainly nothing of the nature of a bubo as described by certain observers.

HEART. Aoyama³, Wilm²³, Lustig and Zardo¹⁷, Fujinami¹¹, Strong, Crowell and Teague^{20, 21} all describe in more or less detail, well-marked

cloudy swelling of the muscle tissue of the wall of the heart. Aoyama², Wilm²³, Lustig and Zardo¹⁷, Fujinami¹¹ and Flexner¹⁰ describe, in addition, fatty degeneration. On the other hand, Albrecht and Ghoni¹, who say that practically no change can be demonstrated microscopically, Hamdi¹³ and Strong, Crowell and Teague observed or record no fatty degeneration of the fibres. Childe⁵ Yamagiwa²⁵, Fujinami¹¹ and Strong, Crowell and Teague lay some stress on the "fragmentation" of the muscle fibres. Hamdi, however, is apparently not dealing with acute plague infection; it is not remarkable to find, therefore, that he notes the presence in the myocardium of a series of indurated nodules composed of fibro-connective tissue. He guards himself, however, by pointing out that as his specimens of muscle had been preserved in alcohol, he may be somewhat rash in stating that there is no cloudy swelling or fatty degeneration of the muscle. Childe, who gives the most complete account of the heart muscle as found in these cases of acute plague, after pointing out that many of the patients die suddenly of heart failure, notes that under the microscope some of the muscle fibres are well stained and distinctly striated, but that others stain very badly, and that in them the striation is "faint or absent, and the muscle substance is swollen, broken up into irregular lumps and of a shiny homogeneous appearance." This condition, he maintains, is associated with dilatation of the heart, and the liver is often in the "nutmeg" condition when examined post-mortem; but from our examination of the liver tissue from which our drawings are made we are inclined to the view that the appearances presented in the liver in these cases are due to acute degeneration of the liver cells around the hepatic veins, and not to a chronic venous congestion. Strong, Crowell and Teague find fragmentation of the fibres of the heart muscle a constant feature in all the cases they examined. There is, then, considerable difference of opinion as to the exact lesions to be found in the heart muscle, but most observers describe dilatation of the right ventricle, especially of the conus (Fujinami), with thinning of its walls and a condition of general cloudy swelling of the muscle fibres of the heart, sometimes with fairly well-marked fatty degeneration. Epicardial ecchymoses are described by most of them, whilst only Strong, Crowell and Teague note a well-marked oedema of the heart muscle especially in the neighbourhood of the fragmented areas.

In the fragment of heart muscle at our disposal, there appears to be some slight oedema of the connective tissue on the surface of the heart, and running between the muscle fibres of the substance of the wall. On the epicardial surface is a distinct haemorrhage, the blood making its way along the lines of the connective tissue between the small collections of epicardial fat. In the oedematous, imperfectly staining, epicardial tissue, and especially between the extravasated blood and the myocardium, are considerable accumulations of polymorphonuclear leucocytes, with here and there a few hyaline cells. Beneath these accumulations, the heart muscle is somewhat oedematous looking, the spaces between the bundles of muscle tissue being of considerable size, the muscle being, as it were, dissected into little bundles, between some of which haemorrhages extend along the lines of the delicate interfascicular connective tissue. At intervals, especially near the surface of the myocardium, are areas in which slight proliferation of the nuclei, either of the sheath of the muscle fibre

or of the capillaries, appears to have taken place. Moreover, this same sheath is considerably swollen and stands out very prominently, forming a kind of yellow, picric acid stained network between the muscle fibrils. It is difficult to distinguish the outlines of the walls of the interfascicular capillaries as the red blood corpuscles appear to be "laked" and to have lost their outline. In many parts of the section, the transverse striation of the muscle fibre is greatly obscured. Here and there are seen vacuoles or collections of fluid actually within the muscle fibres, but no fat granules or globules can be distinguished. Where the muscle is least swollen, the transverse striation can still be made out, though there is marked cloudiness, but where the swelling is pronounced the granularity is lost and the muscle substance has become hyaline and highly refractive. Here also, it is undergoing distinct fragmentation, apparently the result of traction by the more healthy transversely striated fibres upon the swollen hyaline and granular fibres. The fractures in these hyaline masses usually extend right across the muscle, half a dozen of such fractures lying close together. The condition corresponds very closely to the "vitreous degeneration" so fully described by Zenker in the abdominal muscles of patients succumbing to malignant typhoid fever. Commencing multiplication of the intermuscular endothelial cells may be made out very distinctly. Some of these proliferating endothelial cells are highly vacuolated, especially where there is other evidence of oedema—i.e. where the "dissection" of the muscle shows up the cells distinctly. The basement membrane lying between the endothelial cells of the capillaries and the muscle cell is distinctly swollen, even where the yellow tinting is absent.

LIVER. Most writers on the morbid anatomy and histology of plague describe small haemorrhages into the substance of the liver especially just beneath, or into, Glisson's capsule. All agree that engorgement of the hepatic vessels is a marked feature, but Childe⁵ states that engorgement and haemorrhages generally are not so marked in the liver of a patient dying from pneumonic plague as they are in the liver of those succumbing to the bubonic form of the disease. Yamagiwa²⁵ and Wilm²³ mention that the central hepatic vein and the capillaries in the central zone of the lobule are distended with red blood corpuscles. Aoyama³ states that the interacinous vessels of the liver are, as a rule, markedly congested whilst surrounding these vessels and lying in the connective tissue are masses of round cells sometimes in considerable numbers. Wilm describes the liver in these cases as usually large and firm and the blood extravasations as varying considerably in size. He also points out that the boundaries of the lobules are often indistinct. Both these latter observers agree that in a few cases where jaundice has been present during life the liver may be greenish yellow in colour, and that cloudy swelling of the hepatic cells is always a marked feature, whilst Fujinami¹¹, who was able to prepare the tissues for the demonstration of fat, found that the parenchymal cells contain fat droplets when stained with Sudan III. The cloudy swelling may be so far advanced that the nuclei become almost invisible. Hamdi¹³ and Albrecht and Ghon¹ draw

attention to the vacuolated, almost honeycombed, structure of the hepatic cells. They maintain that the vacuoles may contain fat but are unable to demonstrate it in their specimens as they have not been specially prepared. Most authors draw a distinction between pneumonic and bubonic plague in that in the former the so-called abscesses are absent, whilst in bubonic plague they are said to arise as the result of the impaction of emboli in branches of the hepatic artery (Albrecht and Ghon) or of the portal vein (Hamdi). Small necrotic areas surrounded by a haemorrhagic zone are described by Albrecht and Ghon. In these areas large numbers of plague bacilli may be seen, especially in the necrosed centre; at the periphery of these foci are numerous polymorphonuclear leucocytes and red blood corpuscles. Hamdi draws attention to the phagocytic activity of Kupffer's cells and the cells lining the portal capillaries. Strong, Crowell and Teague^{20,21} note that the small metastatic abscesses "occasionally observed in bubonic plague were not encountered in either the liver or the kidney" of cases succumbing to the pneumonic form of the disease. Plague bacilli and fowl-cholera-like bacilli, sometimes in large numbers, are described by Aoyama as present in the inter- and intra-lobular tissue.

The appearance of a section of the liver in which the most marked changes have taken place is, at the first glance, that of chronic venous congestion. The enormously distended central vein of the lobule is, however, always filled with blood in which are small masses of coagulated fibrin usually attached to the wall of the vein, which may be considerably thickened. Around the central vein and in an area corresponding to the central and intermediate zones, the capillaries are greatly congested, and the liver cells are represented merely by imperfectly stained nuclei and extremely granular and vacuolated fragments of protoplasm. Some of the vacuoles may represent fat globules, but they appear to be rather accumulations of clear albuminous fluid, or perhaps even glycogen. It is, however, impossible to determine this in the hardened tissue. The walls of the portal capillaries are somewhat swollen and stand out very distinctly, but neither in the walls of the hepatic vein nor in the walls of these capillaries, nor again in the remaining fragments of liver cells, are there any pigmented granules such as we should expect to find were this a condition of chronic venous congestion. The peripheral portions of the columns of liver cells stand out in marked contrast with the fragmented liver cells of the central zones with which, however, they are seen to be continuous. The whole picture, then, is that, not of a chronic venous congestion, but of an *acute red atrophy*. The hepatic cells at the periphery of the lobule are in a condition of extreme granularity and acute vacuolation, although there is no great increase in the actual size of the cells. The protoplasm of some cells may be simply granular; in others highly vacuolated; in others, again, the nucleus is surrounded by a ring of vacuoles which appears to separate it sharply from the granular protoplasm. In this peripheral area there is very marked swelling of the endothelial

cells lining the capillaries, their nuclei are very distinct and their protoplasm stands out with great sharpness. In the degenerated area the endothelial cells though retaining their outline, stain very badly and losing their nuclear stain appear to be merged into the thickened wall of the capillary vessel. Though numerous fairly well-stained nuclei may be seen, few of them belong to the liver cells, in which the nuclei along with the remaining fragments of protoplasm are very imperfectly stained. Here and there, lying in the vascular channels, may be seen small accumulations of leucocytes or lymphocytes in some of which the nuclei are fragmented. These leucocytes, a few endothelial cells and one or two persisting liver cells, are the only structures in which stained nuclei can be seen, though a number of fragments of nuclear substance, stained and unstained, may be seen. Even the nuclei of the liver cells are, in many places, seen to be undergoing pyknotic disintegration. Contained within the capillaries, growing into their thickened walls and sometimes grouped as though they had been contained in a liver cell, are enormous numbers of plague bacilli, a few of which appear to be adherent to the surface of the Kupffer cells whilst others have been taken into their substance. Except where the liver cells are completely broken down, few plague bacilli are seen in their substance unless they have made their way into the sinusoids of the cells. A very characteristic feature in this condition is a translucent thickened membrane which seems to form a kind of basement membrane between the capillary and the liver cells where the latter are present, and a kind of network where they have disappeared. In the somewhat larger branches of the hepatic vein, the endothelial lining cells are swollen, but their nuclei are still distinct. Here, too, the sub-intimal connective tissue has absorbed a considerable amount of fluid and is greatly thickened; it appears to be in a condition of "hyaline" degeneration. Many of the endothelial cells are detached and are embedded in a clot made up of granular fibrin, a large number of disintegrated red blood corpuscles and a few normal corpuscles. At a few points, taking the place of the disintegrated liver cells, are small accumulations of mononucleated hyaline cells and polymorphonuclear leucocytes. Here plague bacilli are fairly numerous. The large hyaline cells, some of them possibly Kupffer's cells, appear to be undergoing division, but in some of the mononucleated cells the nucleus is dead and in haematein and van Gieson stained specimens the protoplasm of the cell has taken on one shade of yellow and the nucleus simply a deeper shade. The larger blood-vessels, both portal and hepatic veins, are filled with blood clots containing an enormous number of bacilli; many dead or dying leucocytes

(as evidenced by their staining reaction) may be seen, along with a certain number of leucocytes which still retain their staining capacities. The branches of the hepatic artery also contain blood clots, but a smaller number of bacilli, many of them adherent to the lining endothelial cells, which may be detached, are usually granular and often vacuolated, and may no longer take on a nuclear stain. In the small bile ducts, the epithelium appears to separate from the underlying membrane rather more readily (? fluid present) than usual, and there is a curious opening up of the connective tissue spaces in the outer portion of the wall of the duct. Similar distension of the tissue spaces may be seen everywhere in the connective tissue. A finely granular cast, devoid of pigment, occupies the lumen in some of the bile ducts.

The capillary vessels are often separated by distinct spaces from the liver cells; in these spaces very few bacilli are found, though in the capillaries themselves the bacilli may be numerous. These spaces appear to result from oedema, an accumulation of fluid around the capillary and between it and the liver cell corresponding to the similar accumulation in the connective tissue of the wall of the bile duct and around the epithelium within the basement membrane of the bile duct. The coagulation of the blood within the vessels and the formation of ante-mortem clots correspond very closely to the similar conditions noted in the capillary vessels of the lung. Here again, the masses of bacilli are so dense that in an unstained preparation they appear almost like fibrinous clots embedded in the mass of red blood corpuscles, although there is little fibrin present.

In the second liver, in which the changes are less advanced, there is intense congestion throughout the whole capillary system, more marked, however, at one or two points; there is also imperfect staining of the liver cells, with great exaggeration of this feature in areas which are evidently minute necrosed patches, the result of an acute degeneration of a little area of liver cells; there is a slight increase of nuclei, principally of leucocytes, in some of the interlobular fissures and spaces.

In the portal vein are clots, some of which consist of masses of red blood corpuscles, others of homogeneous coagula in which are a few bacilli; in the margins of these coagula and still within the vessel are little groups of leucocytes, between and around which numerous bacilli may be seen. The endothelial cells of the large vessels are exceedingly granular, and at one or two points they appear to be proliferating; on them leucocytes accumulate in considerable numbers. The connective tissue fibrils beneath the endothelium are greatly

swollen and between them leucocytes may be seen pushing their way. Here, as in the larger vessels of the lung, the bacilli are very numerous. In the hyaline-looking clots many "shadow" nuclei—nuclei very imperfectly stained—may be seen lying between the more pronouncedly stained nuclei. These shadow nuclei are swollen and vesicular and show but a slight trace of reticulum. In the congested capillaries, bacilli single or in small groups are present. In these same capillaries are "giant cells" which appear to result from proliferation and vacuolation of the nuclei of a series of endothelial cells, the protoplasm of which has run together to form a protoplasmic mass. Small accumulations of polymorphonuclear leucocytes and of lymphocytes with one or two large hyaline cells may also be found in the capillaries; indeed, taking the section as a whole, these stained leucocytes in the capillaries are considerably more numerous than in a normal liver. The lymph spaces in the connective tissue—which in this case is increased in amount, quite apart from the plague—are greatly dilated as though the tissue were oedematous, but there appears to be some recent proliferation of the cells lying between the bundles of collagenous fibrils. In the vessels of this connective tissue are a few bacilli, but they are by no means such a prominent feature as in the portal capillaries.

Special attention has been drawn to the small areas of badly stained degenerated liver tissue. Here the whole of the liver cells are imperfectly stained and even under a low power the tissues have a curious woolly look, have lost their sharp outlines and their nuclei stain badly. In these degenerating areas, contrary to what would be expected, the number of bacilli is small, only a few isolated organisms being found in some of the clots in the capillaries, although they may be exceedingly numerous in some of the capillaries in the immediate neighbourhood of the dead masses. Throughout the section the hepatic cells are in an advanced state of cloudy swelling; in many cases it is very difficult to make out the nucleus, and where present, it is swollen and vacuolated. There is no evidence of the presence of fat in the swollen granular cells, and as a rule no bacilli can be detected in the substance of the cell, though in some of the preparations there appears to be great dilatation of the sinusoids, whilst the bile capillaries are distended with a clear colourless fluid, and the central lumina of the capillaries stand out much more clearly than they do in a normal liver. Around some of the nuclei in the liver cells is a clear "court," formed by a series of vacuoles within the protoplasm arranged in a circle around the nucleus. This is certainly not due to recent fatty infiltration. Towards the periphery of the lobule the structure can be made out more

easily than nearer the centre and here and there, along with the cloudy swelling, is distinct vacuolation of the cytoplasm of the hepatic cells corresponding apparently to the vacuolation seen in the parenchymatous cells of livers taken from cases succumbing to other *acute* infective fevers though this *may* indicate an earlier fatty infiltration of the liver cells. The granular and vacuolated structure of the liver cells, the disintegration by fragmentation and the imperfect staining of the nucleus, are very well brought out by the haematein and van Gieson stain, as is also the vacuolation of the nucleus; in some cells several vacuoles are seen, in others a single large vesicle with fragments of chromatin pushed to the periphery. The cytoplasm of many of these cells resembles a mass of foam. One or two bacilli may be seen apparently invading the sinusoids, and even in the bile ducts, but this is not of frequent occurrence.

In the connective tissue in the portal spaces as in other new connective tissue, the oedematous condition is well marked, large clear spaces, in which few bacilli can be seen standing out very prominently. Here too and around the bile ducts there is a slight increase in the number of connective tissue nuclei. The capillaries are enormously dilated.

Where the epithelium usually lining the capillaries has been detached, bacilli appear to be adherent to a kind of basement membrane. The endothelial cells of the capillaries are often imperfectly stained, their nuclei are obscured and badly stained, their protoplasm cloudy and vacuolated. Sometimes, however, the endothelium still stands out very distinctly. Here again the hyaline clots appear to contain an enormous number of plague bacilli embedded in a clear homogeneous coagulum. In the immediate neighbourhood of these clots the red blood corpuscles run together, forming hyaline, orange yellow masses. The blood platelets and masses of red corpuscles in these hyaline clots undergo very considerable modification as regards their staining, and instead of an orange yellow they take on a distinct pink tinge, swollen and either single or in hyaline masses they form a very characteristic feature. They appear to have been acted upon directly by the fluids in which the bacilli are multiplying. In this case the multiplication may be post-mortem, as this liver has not been so well preserved as have most of the other specimens. No fibrin is to be seen in any part of the section. Many pairs of nuclei are seen in Kupffer's cells near the margin of the lobule, where there is a great accumulation of proliferating endothelial cells.

SPLEEN. There seems to be some little difference of opinion as to the changes that take place in the spleen. Childe⁵, for example, states that in the pneumonic form of plague, the spleen is less engorged and

has fewer haemorrhages than the spleen taken from patients suffering from bubonic plague; whilst Flexner¹⁰, Aoyama³ and Wilm²³ all describe the spleen as being greatly enlarged, from two to five times the normal size, deeper red in colour, the pulp swollen, soft, hyperaemic and oedematous (Flexner). Sometimes, according to Wilm the pulp may be harder than usual. Haemorrhages under the capsule and into the pulp tissue are described by Childe, Aoyama, Wilm, Lustig and Zardo¹⁷, and Strong, Crowell and Teague^{20,21}, who point out, further, that the congestion is specially well marked at the immediate periphery of the lymphoid follicles. The Malpighian bodies are said to be affected differently in the two forms of plague. Flexner, Aoyama and Wilm describe them in the bubonic forms as being enlarged or hyperplastic, both as regards endothelial and lymphoid cells, through multiplication of the cells of the lymphoid cords or follicles (Malpighian bodies), whilst Strong, Crowell and Teague maintain that in the pneumonic form these lymphoid follicles are for the "most part both relatively and absolutely small and seldom show any signs of proliferation." The latter observers say that the bacteria in these follicles are scarce and that, although there is swelling of the endothelial cells, there is little evidence of multiplication. Flexner describes hyaline changes in the walls of the arteries of the Malpighian bodies, and a cellular proliferation in the sub-intimal layers of the veins. He also describes leucocytes attached to the inner wall of the vessel, sometimes appearing to cause detachment of the endothelial cells; rarely is there any phagocytosis. Bacilli, he says, occur specially in the pulp, attaching themselves to the reticulum, and growing in irregular masses into the venous sinuses; they may occlude small blood-vessels and lymph spaces, and are so numerous and accompanied by so little reaction of the tissues that he is led to suggest that many of them have made their appearance after the death of the patient. Flexner, Yamagiwa²⁵, Albrecht and Ghon¹, Hamdi¹³, and Strong, Crowell and Teague all describe necrotic patches, the latter who describe a small reddish-white infarct 4 mm. in diameter, associating this condition with the small haemorrhages that are here met with. Albrecht and Ghon, however, maintain that the source of the histological changes in these foci lies in the infiltrating cells, as, under their influence, necrosis of the wall of the capillary vessel takes place, often with a peculiar running together or coagulation of the tissue of the walls of the blood-vessel, of the blood and of the breaking-down cells in the immediate neighbourhood.

In our case the spleen was acutely congested, and the Malpighian bodies, though relatively smaller, were actually larger than normal. They stood out fairly distinctly from the deep red background of the pulp tissue. On microscopic examination, the fibrous trabeculae and the capsule are swollen and oedematous looking, clear spaces appearing in all the fibrous tissue. The central artery of each appears to occupy a greater area than usual apparently because of the great hyaline swelling of its walls, especially the intima, in which are large vacuoles. The endothelium lining the vessel is swollen, cloudy and somewhat imperfectly stained. Around, and in the immediate neighbourhood of these vessels, large open spaces, evidently filled with clear fluid, give the adventitia a very characteristic appearance. The reticulum of the adenoid tissue stands out very prominently, its strands are swollen and the meshes of the network enlarged; the lymphocytes are numerous—more numerous than usual—but do not crowd or

fill up the reticulum completely, and the whole tissue looks much more open than normal adenoid tissue. Few red blood corpuscles are seen in the vessels of the Malpighian body, except at its extreme margin. As soon as the pulp tissue is reached, however, the sinuses are seen to be enormously distended, and the usually delicate reticular framework of the pulp now stands out as thick hyaline or slightly granular bands lying between masses of red blood corpuscles. The sinuses, both arterial and venous, are greatly enlarged and filled with red blood corpuscles, a few detached endothelial cells, and a number of polymorphonuclear leucocytes. The endothelium lining the spaces is distinctly swollen, often vacuolated, and at points even proliferating. The nuclei of these endothelial cells stain imperfectly. Around the nucleus there may be little groups of vacuoles similar to those seen in other cells, the nucleus being cut off, except by very fine strands, from its cell protoplasm. The swollen strands of the reticulum appear to have absorbed a large amount of fluid. So-called giant cells (little more than a mass of cells, of which the protoplasm has run together, but not the nuclei) are seen. No fibrin can be made out in any part of the spleen.

There is pyknotic degeneration of the nuclei of some of the cells lying in the sinuses, especially where the bacilli are numerous. The walls of the smaller nutrient arteries are distinctly hyaline. In the central vessel of the Malpighian body the nuclei of the endothelial cells are very badly stained and distinctly vesiculated. Vacuoles appear in the connective tissue of the intima between the fibrils and in the nuclei of the cells, but here no plague bacilli are seen. Many of the lymphoid cells lying in the reticular spaces of the Malpighian body contain vacuoles. As we come near the margin of the adenoid sheath, a few bacilli are found; and then in the pulp an enormous number of plague bacilli may be seen lying on the swollen trabeculae, especially when the sinuses are reached. Here a number of bacilli are seen lying free, sometimes in a hyaline-looking material similar to that seen in the blood-vessels of the liver and lung but now and again isolated. Here also some of the red blood corpuscles appear to be so altered that they take on a pink tinge; this, however, is not nearly so marked as in the intravascular coagula in the liver. Small areas of badly stained tissue—focal necroses—appear wherever large numbers of bacilli are concentrated, but how far this is a post-mortem condition it is difficult to say.

The most interesting features presented are the swelling and vacuolation of many of the endothelial cells in the sinuses, the bacilli making their way from the lumina of the larger

vessels into a softened swollen intima, in which there appears to be cell proliferation and infiltration with polymorphonuclear leucocytes, and the pyknotic changes where the bacilli are numerous; the invasion of endothelial cells by large numbers of bacilli, the strings of bacilli adherent to the walls of the sinuses where the epithelium has been detached; and the pyknosis and extreme vacuolation of these separated cells.

In addition to these, large marrow-like giant cells which do not take up other cells or bacilli, others, several times the size, which appear to have ingested degenerated leucocytes but no red cells, may be found in the clots in the sinuses. Some of these also contain bacilli.

In the small Malpighian bodies the bacilli appear to be more numerous, especially where the blood-vessels and capillaries are dilated, but they are never so numerous as in the pulp tissue.

KIDNEY, Childe⁵, reporting on cases of pneumonic plague, mentions that the kidney, like other organs, resembles the corresponding organ taken from cases of the bubonic form, "except that the general engorgement and haemorrhage are less marked"; that large haemorrhages are usually absent, but that engorgement and petechiae may be found in the pelvis of the organ; that it is enlarged, the capsule stripping off easily, and showing petechial haemorrhages in the pale, soft, degenerated parenchymatous tissue beneath. The venae stellatae are very visible (Aoyama³). The cortex appears to be somewhat thickened (Aoyama, Wilm²³). Strong, Crowell and Teague^{20,21} describe the outer surface of the kidney after removal of the capsule as presenting a red granite-like appearance due to the standing out of the deeply injected vessels against the pale grey or yellowish parenchyma of the organ. They also mention that "the glomeruli were frequently swollen and often appeared as fine, reddish, pin-point-size areas. Petechiae were frequently seen in the pelvis and upper portion of the ureters." Although this is not explicitly stated, these observers evidently noted swelling of the cortex.

Most observers seem to agree that after congestion, cloudy swelling of the epithelium of the convoluted tubules is by far the most common histological feature presented in the kidney. Some cloudy swelling of the epithelium in the straight tubules is also described. Fatty degeneration also occurs—Aoyama, Wilm, Strong, Crowell and Teague, and Fujinami¹¹. Hamdi¹³ describes the contents of the urinary tubules as "mostly a reticular coagulated mass and hyaline cylinders," resulting from breaking up of the protoplasm of the degenerated cells. The nuclei in these cells stain very imperfectly (Aoyama, and those who describe cloudy swelling). Yamagiwa²⁵ mentions that the lumina of many of the urinary tubules are plugged with granulated cylinders, but Aoyama and Hamdi describe hyaline cylinders in this position, and Strong, Crowell and Teague a coagulated fluid exudate or transudate, as being of not infrequent occurrence. Unchanged blood or red blood corpuscles are also said to be present in the urinary tubules, often accompanied by clumps of bacilli. Most of those who have made careful examination of the kidneys in these cases of pneumonic plague, describe changes in the glomeruli, and it is evident that both in bubonic and pneumonic plague the alterations are due to a secondary septicaemia, rather than to any peculiarity of the type of plague that is under consideration. Engorgement of the vessels of the glomeruli is always present; and

apparently coagulation of the blood, accompanied, possibly, by a coagulative necrosis of the cellular walls of these vessels, takes place, this corresponding to the condition of cloudy swelling or granular degeneration met with in the epithelium of the convoluted tubules. This coagulation of the blood is specially insisted upon by Aoyama and Albrecht and Ghon¹, who mention that the coils of the glomeruli may, in places, be homogeneous, vitreous, and dilated; and that they may be transformed into bands, which consist of trabeculae or thread-like coagulations,—indeed, they describe a coagulation not only of the tissues, but of the fluids of the tissues, and of the vessel elements themselves. Aoyama describes a multiplication of the nuclei of the glomeruli, but Strong, Crowell and Teague pass over this and insist rather on the degeneration of the epithelium covering the glomerular tufts, following Aoyama in so far that they describe a fluid exudate as present in Bowman's capsule and also in stating that this space is often filled with desquamated cells or with a granular exuded mass. Strong, Crowell and Teague are the only observers who confirm Herzog^{13a} in describing fibrin thrombi as occurring in the glomeruli. Hamdi states that the changes in the interstitial tissue of the kidney are slight, but Aoyama, and Strong, Crowell and Teague mention a separation of the connective tissue fibres and put it down to an oedematous condition. The vessels in this interstitial tissue are described by them as "greatly dilated." All observers who mention the bacilli at all, describe them as present in the interstitial tissue, in the glomerular vessels and in both convoluted and straight tubules, especially in the latter, where the amount of blood is usually greater than in the convoluted tubules.

We found the capsule of the kidney slightly thickened, apparently the result of separation of the laminae by oedematous fluid. The walls of the arterioles are somewhat thickened and hyaline, many of them are distended with clot, made up partly of red blood corpuscles, partly of the same hyaline material seen in the vessels of other organs; the intima is thickened and hyaline. The Malpighian bodies are considerably enlarged, owing (1) to congestion of the capillary loops, and (2) to a thickening of the walls of these vessels and of the tissue between them, in which there seems to be some slight increase in the number of nuclei. Even with a low magnification, a homogeneous looking exudate, partially filling the space between the glomerular tuft and Bowman's capsule, is seen in some of the Malpighian bodies. Throughout the section, the basement membrane of Bowman's capsule is swollen and looks as though it had absorbed fluid; the well-preserved endothelial lining of the capsule composed of an almost continuous layer of swollen cells has separated from the swollen capsule.

The nuclei lining Bowman's capsule are very distinctly stained and appear in some cases to be undergoing division, as do also similar cells covering the capillary loops. In the capillaries in the tuft a few bacilli may be seen adherent to the endothelial wall. The clot in the large vessels consists of a granular mass, in which may be seen colourless shadows of red blood corpuscles. Examining one of the clots carefully, a large mononuclear leucocyte with imperfectly stained nucleus

and a large vacuole in the protoplasm may be seen in the centre and scattered through it are some half dozen isolated bacilli. In the intertubular capillaries the congestion is very marked. The endothelial cells lining these capillaries are often swollen and vacuolated, and the basement membrane thickened and prominent. In the finer capillaries, groups of bacilli may be seen, usually adherent to the endothelial wall. In the capillary loops, owing to the swelling of the basement membrane, the intima has a much coarser appearance than in the normal Malpighian body. Wherever there is exudation in the Malpighian body, the cells lining Bowman's capsule show a large nucleus, and the cytoplasm is granular and swollen. The nuclei of a few of these cells take on no nuclear stain; they are of the same tint as the cell protoplasm. The changes in the epithelium of the convoluted tubules are very characteristic. In a few tubules, there is simply enormous enlargement and great granularity of the cytoplasm of the large epithelial cells; in others the granular protoplasm is distinctly vacuolated; in others again there appears to be little more than a network of granular protoplasm, with, here and there, a nucleus more or less altered, whilst in some of the tubes there are simply a few granules and a few nuclei to which are attached minute portions of cell protoplasm. All this results apparently from the extreme swelling and vacuolation of the cells, followed by rapid disintegration of the cytoplasm. Although the bacilli are fairly numerous immediately under the basement membrane of these tubes—that is, in the capillaries—we were unable to find any bacilli in the tubules in which this rapid and extreme disintegration was going on. Careful search was made for the presence of red blood corpuscles in these convoluted tubules, but none were observed; they certainly cannot be numerous even in the lower part of the collecting tubules. In the looped tubules of Henle, the more or less cubical epithelium is but little altered. In the collecting tubules there is cloudy swelling of the cubical epithelial cells, or even some disintegration of the protoplasm. In these tubules, too, are little collections of granular golden-brown pigment, which can only be derived from red blood corpuscles. Here again there is marked swelling of the basement membranes of the tubules, whilst the nucleus of the epithelium is, in a large number of cells, almost surrounded by a free space, though very delicate threads may be seen passing from the margin of the nucleus to what looks like a delicate cell wall. These are evidently “dropsical” cells which ultimately break down, and only a few granules and the free nucleus above described remain. In the lower part of some of the tubes, the nuclei with small fragments of cytoplasm attached, have accumulated and formed casts

which might easily be taken for giant cells, but they are distinctly within the tubes. The degeneration of the endothelial cells lining Bowman's capsule appears to take the form of great enlargement, cloudy swelling of the protoplasm, and impaired staining of the nucleus. Even in some of the connective tissue cells in the glomerular tuft, vacuolation is observed around the nuclei of the connective tissue or endothelial cells. It is rather extraordinary that with such intense congestion there should be so little evidence of haemorrhage into the tubules. Here and there hyaline casts may be seen. In one or two of the medium-sized veins, as in the veins of other organs, one has an almost pure culture of the plague bacillus.

GENERAL REMARKS

The histology of these specimens appears to us to be of special interest in that it affords evidence of the presence of an extremely acute septicaemic condition. The heart, with the vitreous condition of the muscle and the comparatively slight degree of cellular proliferation, has evidently been affected by very active toxins. In the liver, we have typical examples of the lesions produced by specific infective micro-organisms that give rise to toxic substances; the swelling of the parenchymal cells, the cloudy swelling, the vacuolation, the rapid disintegration of the cytoplasm, the vesiculation of the nucleus and the hyaline swelling of basement membranes all pointing in the same direction. The epithelium of the kidney, granular, vacuolated and disintegrated, and with its altered nuclei, is modified by a similar toxic activity, the toxin being excreted by the epithelial cells and in a lesser degree in the fluids that pass out from the capillaries of the glomerular tuft into the capsule. All the basement membranes are swollen. In the spleen, the principal changes are in the walls of the central artery of the Malpighian body, in the reticulum of the highly-developed adenoid sheaths of this vessel, and in the lining endothelium and basement membranes of the splenic sinuses. In all these positions, rapid and marked changes have taken place, such as would in any other infective disease at once be ascribed to a septicaemic condition.

In the lungs, the lesions are far less marked than one would expect, were the pneumonia the main or most important factor in the disease. It is obvious, of course, that the pneumonia is lobular in type and that it differs very materially from the pneumonia set up by the *Diplococcus pneumoniae* which was never found in any section stained by Gram's method. The greater part of the lung tissue is oedematous, a condition which, from the histology and from the symptoms as described by those who examined the patients in the epidemic during

which these specimens were collected, appears to have arisen at a later date than the catarrhal pneumonia, though this may not apply to the bronchitis, a characteristic feature in the case under examination. It would, of course, be foolish to found any general argument on observations made on one or two cases, but it appears to us that here at any rate there has been an acute bronchitis, induced, perhaps, by a large dose of the plague bacillus, and that along with this, there has been some collapse and catarrhal pneumonia, especially as in the immediate neighbourhood of the collapsed and catarrhal patches, some of the alveoli are distended and emphysematous. From these catarrhal pneumonic patches, the *Bacillus pestis*, the exciting cause of this local disease, has passed into the lymphatics and so to the capillaries in the alveolar walls, and a septicaemic condition has been set up. Following this, the bacilli have been inhaled from the bronchial tubes into other alveoli, and there multiplying, have not set up any cellular reaction, but have induced a marked oedema, the bacilli with their poison in the circulating blood counteracting the positive chemiotaxis and cell reaction that would otherwise have occurred in the air vesicles. We have, in fact, a repetition of the conditions met with in malignant oedema, when the malignant oedema bacillus is injected both into the muscle substance and into the vein. This, we think, accounts, in the case under review, at any rate, for the lack of coagulated fibrinous lymph, not only on the surface of the lung, but also in the alveolar spaces; it also accounts for the lack of fibrin in the other organs, the septicaemic condition interfering with the passage of leucocytes from the vessels. The exudate resembles that of passive oedema rather than that of an acute inflammatory exudation.

Our investigations seem to us to point to the suggestion that in the Manchurian outbreak the amount of the infective material gaining access to the upper respiratory passages, to the tonsils, fauces, etc., is of prime importance in determining the character of the septicaemia—for we are satisfied that this is a septicaemia or bacteraemia resulting from a pulmonary infection. In warm countries where the people live in the open and where the facilities and channels by which infection is communicated appear to be those provided by rats and fleas, and where the plague material is carried more or less directly from one patient to another, or from the rat, by the flea, to the human subject, the local reaction of the tissues and the bubo may prevent the extension of the bacteria, especially if the dose be small and the septicaemic condition occurs at a comparatively late stage; whilst if there is good reaction of resisting tissues, the disease may never become septicaemic.

If, however, the septicaemia once develops, it is evident that the internal organs are affected much as in the pneumonic form of plague. In Manchuria, where plague was at its height in the depth of winter, the facilities for the inhalation or ingestion of large numbers of plague bacilli were far greater than they can possibly be in warm countries where people live "in the open." The patients, residing in badly ventilated houses, closed because of the intense cold, and heated artificially, are living in highly infected incubators of the most approved kind. Septicaemias are rapidly developed, and although the lung might in a certain proportion of cases be the primary seat of infection, the late symptoms—smaller or larger areas of dulness, rales, only shortly before death (if coarse rales are heard it is looked upon as evidence that the patient is not suffering from pneumonic plague), oedema, bacilli in large numbers in the sputum—observed seem to point to the occurrence of a secondary pneumonia in a certain proportion of the cases examined clinically. To explain the differences of opinion as to the importance and meaning of certain histological findings is difficult, though it may be suggested that the pleuro-pneumonia with a fibrinous lymph observed in certain cases may indicate the presence of a primary pneumonia in which there has been a massive infection by massive doses, whilst the catarrhal pneumonia with bronchitis followed by oedema appears to suggest a pneumonic condition following a septicaemia. Should we be able to obtain more material for examination, we hope to return to this question.

Although it is probable that the *Bacillus pestis* multiplies in various sites, but especially in the vessels, after death, and even whilst the patient is dying, it is evident that during life there must be an enormous multiplication to produce such a virulent septicaemia as that indicated by the lesions observed. We cannot but feel that during the earlier stages of the disease the tissues and fluids of most patients must exert a very active bactericidal action, and that the disease was so intensely fatal in the Manchurian epidemic and the patients succumbed because the dose of infective material was always massive and entered by ever open and slightly resistant portals. It may very well be that during an epidemic of bubonic plague a considerable number of patients may be actively immunised by small doses of bacilli introduced by bruises or punctured wounds however made and infected. This, we think, must account for the sharp line of demarcation, as regards susceptibility, that exists between patients living under conditions unfavourable to massive infection and those living under conditions in which they are exposed more or less continuously to massive doses of the infective agent. It would appear that when the massive dose

has been taken, the prognosis is bad, unless in the meantime there has been some reaction of the tissues as a result of which an active immunity has been gradually developed. In the case of the bubonic plague, the advance of the bacillus is so interfered with that not only a local, but a general immunity may be acquired before the bacilli can reach the blood in any considerable numbers, whilst in the case of infections by the lungs or naso-pharyngeal regions, the local reaction and resistance being comparatively slight and the dose of infective material large, the septicaemic condition is very rapidly produced, in many cases before there has been time for the development of any but the earlier typical changes associated with pneumonia.

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THE SECOND PNEUMONIC PLAGUE EPIDEMIC IN MANCHURIA 1920-21

A General Survey of the Outbreak and Course

BY WU LIEN-TEH

- A. Geographical.
- B. General description of the outbreak.
- C. Individual description of infected localities.
- D. Approximate expenditure of anti-plague work.
- E. Chinese refugees from Urga.

A. GEOGRAPHICAL

Transbaikalia or Zabaikalia is a vast stretch of land lying to the north-west of Manchuria, bounded on the east by River Argun from China and on the west by Lake Irkutsk and Irkutsk Mountains from the Irkutsk region. Its most important cities are Chita (capital), Stretinsk, Nerchinsk, Kiakhta and Borzia. The region around Borzia for a hundred miles is composed of undulating hillocks inhabited by a species of large (cat size) marmots or tarabagans (*arctomys bobac*) whose skin is much sought after for the manufacture of imitation sable. The Russians and Buriats find the flesh of these animals wholesome, and often hunt them for the meat and the fat which they use for greasing shoes. It is among the trappers and eaters of tarabagans that the early cases of bubonic plague are usually reported. Manchuria, which receives the butt of the attack whenever an epidemic of pneumonic plague arises, is known by the Chinese as Tung San Sheng (Three Eastern Provinces). It is the largest and easternmost part of China, containing an area of 363,000 square miles and a population at present of 22 million. Its three constituent provinces are:—

Fengtien (*capital* Fengtien or Mukden)

Kirin (*capital* Kirin)

Heilungkiang (*capital* Pukuei or Tsitsihar)

Of these, Heilungkiang Province adjoins Transbaikalia, the town of Manchouli being situated only a few miles from the boundary. The large province of Mongolia touches all three parts of Manchuria on the west. The country around Hailar,

mostly Mongolian territory, is also a tarabagan infested region, and hence Manchouli and Hailar are the two principal skin-collecting depots in Manchuria, and any invasion of plague from the endemic regions is first reported in these two towns.

Beyond the regular caravan and cart routes, there are no proper macadamised roads in the rural parts of Manchuria. The Chinese Eastern Railway begins at Vladivostock (seaport of Maritime Province of East Siberia) enters Manchuria at Pogranitchnaya (Suifenhö), traverses Kirin and Heilungkiang for 886 miles and leaves Manchuria at Manchouli Station. From Harbin (largest city of Manchuria), the Railway branches southward for 150 miles to Changchun (Kirin), where it joins the main South Manchurian line going to Dairen (436 miles). Other railway lines in or adjoining Manchuria are:—

- a. Ussuri R. from Nikolsk to Habarovsk (410 miles)
- b. Kirin-Changchun R. from Changchun to Kirin (77 miles)
- c. Chengchiatun to Szuping kai (55 miles), branch of S.M.R.
- d. Tashihchiao to Yinkow (14 miles) branch of S.M.R.
- e. Peking-Mukden (523 miles)
- f. Mukden-Antung (171 miles)
- g. Antung to Seoul (310 miles) and Fusan (591 miles).

The principal seaports of Manchuria are Dairen and Newchwang (Yinkow), which have regular steamer services with Chefoo, Lungkow, Tsingtau, Weihaiwei (Shantung), Tientsin (Chihli), Shanghai, Canton, Hongkong (South China), as well as with different ports of Japan and Korea.

All these data have an important bearing upon any outbreak of Pneumonic Plague in Siberia or Manchuria.

B. GENERAL DESCRIPTION OF THE EPIDEMIC

For ten years, that is, since the great Plague of 1910-11, Manchuria had been free from the pest, although almost every year, in the intervening period, sporadic cases were reported in some part of Siberia or Mongolia. For instance:—

In 1911 Bissemsky registered 5 bubonic cases at Scharasone.

In 1912 Haffkine reported 3 pneumonic cases at Chita.

In 1913 some bubonic cases were reported in Kirgiz Steppes.

In 1914 we recorded 16 bubonic cases (13 fatal) in Transbaikalia.

In 1917 we recorded bubonic cases at Patsebolong (Mongolia) resulting in the Shansi outbreak and 16,000 deaths.

In 1919 we reported 2 bubonic cases (both fatal) at Ikievskaya.

In 1920 (August) 6 bubonic cases (5 fatal) occurred at Abagatui (5 miles) 3 at Dauria (40 miles) and 4 at Kailastu (50 miles).

In the following October, the wife of a Russian guard stationed on the Railway bridge at Hailar (117 miles from Manchouli) died of bubonic plague. Three out of five sons were infected and died, her husband was also infected but recovered in hospital. These cases were seen at the Railway Hospital and found to be purely bubonic.

Three Chinese soldiers living in the same compound also died of the disease. Owing to the free intercourse between the sick and some outside workmen, the infection was distributed in the city, and efforts at segregation met with opposition from the soldiers. During our stay at Hailar, we noticed the gradual evolution of the plague from the bubonic through the septicemic into the pneumonic form, due principally to promiscuous spitting and huddling together of coolies day and night in unventilated inns. Both here and at Dalainor later on, it was common to find all the inmates of one house numbering 4-8 or more dead of the plague within a few days. On December 12, the Chief of Police at Hailar (Lang), a keen officer was waylaid by soldiers when driving with our Medical Officer Tang, and had his scalp opened by a knock on the head. In spite of this, our record at Hailar was good for only 52 cases were altogether reported. Nine contacts then under observation in a large inn were set free by the soldiers and escaped by rail to different parts. Two reached the coal mines of Dalainor (100 miles), where they infected others in the crowded underground dwellings and thus really started the great epidemic of 1920-21. More than 1000 out of 4000 Chinese miners died of the pest in this Dalainor district as a consequence. From Dalainor, persons incubating the disease escaped to Manchouli (20 miles away) where they were responsible for the deaths of 1141 people including 334 Russians. Contacts also reached Tsitsihar, where 1734 persons lost their lives, and also Harbin, where the deaths totalled 3125. Other towns on the Chinese Eastern Railway received the infection to a greater or lesser extent. Thanks to the rigid measures adopted at Harbin in controlling the regular passenger traffic, limiting the daily sale of third class tickets for each train to 50, medical inspection of all trains arriving and leaving, and quarantining for 5 days of third class passengers at Changchun (150 miles), the epidemic was practically stayed south of Harbin, and only 77 cases were reported altogether at Changchun and 4 at Mukden, as compared with at least 5000 in each city during the epidemic of 1911. The regular passenger traffic between

Manchouli and Harbin was stopped on February 1st, but leakages occurred all along the line (584 miles), and many cases filtered in on freight cars and by road from the infected regions. The addition of a sanitary car to each passenger train perhaps helped in isolating cases found travelling with other passengers. Because of its size and commanding position, Harbin, in 1921 as in 1911, soon became the central focus of infection, but thanks to our early preparation we never lost control of the situation. In spite of the annoying opposition from soldiers and ignorant populace we succeeded in narrowing the spread of the epidemic to every possible limit. Beyond Changchun, very few plague cases were reported in Manchuria, such as 4 at Kungchuling (picked from South Manchurian trains), 4 at Mukden, 18 at Koupangtsu (all traced to one family). A limited outbreak in south Chihli and Sangyuan (Shantung) costing nearly 200 deaths, including that of Dr. Yu Shu Fen (of our temporary staff) was believed to arise from two undetected Tientsin cases (Jan. 22). The port of Chefoo was infected from Vladivostock, but all these instances will be described under separate headings. From Harbin, the epidemic travelled eastwards about middle of February, invading most of the stations of the Railway, and finally reached Vladivostock, where the first case was reported on April 9th. In this city the epidemic raged throughout the summer months and did not terminate until the following October, after causing the death of 520 persons. Altogether the 1920-21 epidemic claimed 9300 lives (including 600 Russians).

The rise and decline of the epidemic form an interesting study. As seen above, the first sporadic (bubonic) cases commenced in August 1920 in different parts of Siberia, e.g. Abagatui, Dauria and Kailastu. In October bubonic cases appeared at Hailar, the Russian woman who first caught the disease having made frequent visits to Manchouli near the Russian and Mongolian borders. Practically her whole family was attacked, only two out of five sons escaping. The outbreak was practically confined to this town during the months of November and December. After the attack upon the Chief of Police, cases first appeared at Dalainor early in January, and then rapidly multiplied. Tsitsihar reported its first case on January 18, the number rapidly increasing. It in turn infected other neighbouring cities. Harbin received its first case from Dalainor—an escaped sick miner, who was diagnosed by us to be true plague on January 22. The highest mortality point was reached in Harbin on April 3. The last case in Manchuria was recorded before end of May. With the last Vladivostock case in October, the second Manchurian plague epidemic may be said to have lasted exactly one year. In 1910-11, the first case in Transbaikalia was also recorded about August. Harbin reported its first case in November and its last in the

first week of April, but the epidemic stayed on in Mukden until middle of May. On that occasion, Vladivostock was not invaded, but several cities in Chihli and Shantung suffered badly, including Peking, Tientsin, Tsinanfu, Tehchow, etc. Strange to say, the two busy cities of Tongshan (coal mining centre 80 miles from Tientsin) and Yinkow (large Manchurian seaport), which used to be centres of bubonic plague arising from cases in Hongkong, entirely escaped in 1911 and 1921.

C. INDIVIDUAL DESCRIPTION OF INFECTED LOCALITIES

1. *Hailar.*

Hailar being the first seat of infection that our staff visited during the 1920-21 epidemic deserves special attention. It is an interesting town, formerly in Mongolian territory. It consists of three parts: (a) railway settlement including the station and containing about 2000 Russian inhabitants, (b) Mongolian area comprising the famous horse market, and (c) Chinese city, seat of the Taoyin of district. The total population is about 10,000. Since the Bolshevik troubles (1918), the Taoyin's rank has been raised to that of a Director-General (Tupan). At the time of my first visit to Hailar (24 hours by rail from Harbin) on November 28th, 1920, the station area was filled with hundreds of cars, thousands of Russian soldiers and their ponies under Kappel and Semenoff, who had just been driven away by the Bolsheviks in the west. It was fortunate that these were sent away within three days', otherwise, some of them might have got infected by the plague and thus spread it broadcast.

It is always difficult to trace the origin of the early cases of an epidemic, but it was particularly so in a wild place like Hailar inhabited as it is by Russian, Chinese and Mongols. As far as I could ascertain, some suspicious deaths occurred on 4-7 October among seven Chinese coolies working in a small skinning factory situated a mile away on the northside of the railroad. Where these men came from, it was impossible to find out beyond the fact that they had something to do with skins. Their bodies were all voluntarily cremated in the compound occupied by them. The second group of cases was carefully studied by me on the spot. These began with a Russian woman Tarelkin (died Oct. 22) who was the wife of the Russian signalman on duty at the Hailar Railway bridge. Besides herself and her husband, there were five sons and twenty-one Chinese soldiers living in the same compound. Of these, her husband, three boys, and three soldiers caught plague, all dying except the husband. The last death occurred on November 25, i.e. 34 days after the first infection occurred in the compound. During my investigations, I attempted to secure some rats and fleas from the infected houses, but failed.

There was no doubt, however, as seen from the list of cases, that they were of a bubonic type, some being treated in Hospital. The only case that recovered was old Tarelkin, aged 43 years, whose bubo in the left groin was opened.

The following information was supplied me by the soldiers on Nov. 29th, 1920.

21 Chinese soldiers, for duty at the Railway bridge, were living in two big rooms in the Tarelkin compound. They had a mutual kitchen shared by the Tarelkin family, the Russians moving freely among the Chinese. The Russian woman used to make regular visits to Manchouli (115 miles away), the last one being on August 13. She showed signs of plague on Oct. 22, and died the next day. Her fourth son, aged 9 years, got sick the same day and died also on Oct. 23. One soldier Chen had fever and rash on body on Oct. 23 and died two days after. After the woman and her 9-year-old son died on Oct. 23, Tarelkin and 4 other sons removed to the same room occupied by sick Chen and stayed there for 5 days. No further sickness was reported for 24 days between Oct. 25 and Nov. 15. Then two Tarelkin boys, aged 18 years, became sick, were admitted to hospital with fever and buboes, and died—one on 17th, the other on 21st. A soldier Chang (aged 19) became sick on Nov. 17 with bubo in left axilla and died on the 19th. He was living with five other soldiers, though he used a separate wooden bed. One of these contacts, Chao, developed plague on 21st, was admitted into hospital on 24th with bubo in left axilla, and died the following day. It was acknowledged by the soldiers that rats were present in the house, and they were frequently bitten by fleas, but they did not remember seeing any dead rats lying about. Besides their three dead mates at the Tarelkin compound, there were perhaps fifteen other soldiers who had died suddenly in Hailar town about that time.

On November 13, one man was reported to have died at Yi Shun Inn after one day's illness. On Dec. 2nd another sick man was admitted into Hospital from another Chinese inn (Yu Tai) with signs of plague and died after 14 hours. At autopsy, both these cases were reported to be septicemic plague. From this date onwards, the cases seen were few and far between, but whether alive or dead, buboes were rarely seen. In their stead septicemic plague was recorded at P.M. On Dec. 8th, a coolie was found dead of plague in an inn, which was then closed by the Police. Nine contacts were isolated. It happened that this inn was partly owned by a local soldier, who resented this interference with his business. He thereupon collected twenty others, and on Dec. 12th, waylaid the Chief of Police Lang while driving in a carriage with our Medical Officer Tang. Lang received a severe scalp wound with much bleeding, and the nine contacts were set free. Telegrams were dispatched by us to the Governor of Heilungkiang, and the Police who had up to now done their work splendidly, refused to go out again. The anti-plague work came to a standstill for one week, during which no precautions were taken. Eventually, Police Chief Lang was prevailed upon to resume duties, but the plague had spread to other regions. The two ringleaders of the soldiers were tried by court-

martial and shot, while their captain—a wild, uneducated man, who at an anti-plague meeting publicly accused the doctors of having brought the plague, was fined and dismissed from the military.

Examinations of passengers buying tickets was commenced on December 18. Inspection of departing and arriving trains continued throughout.

One rat was found on December 23, and autopsied with negative results. After December 20th, cases with cough and bloody sputum were often seen. One report said that a man infected three out of four others in the same house. All the sick died and showed at postmortem congested trachea and bronchi with plague bacilli but no evident pneumonia. Four patients seen on December 27 had pain in front of chest and epigastrium, dyspnoea, no headache, light cough, bloody sputum. On 28th December, an excited man rushed into the Police Headquarters and demanded that all anti-plague doctors be sent away from Hailar; the plague would then automatically disappear! After the punishment of the soldiers, our work became considerably easier, and a total of 52 cases was reported. The question of a sanitary cordon was raised and given up at that time. No more plague was reported at Hailar for some time (Jan. 31st.-Feb. 23) until some reinfections took place from Manchouli and Dalainor. This second invasion was, however, confined to the station area. Drs. Chun and Hsieh reported necropsy of a case seen alive on January 15 with no cough or blood-spitting. The spleen was small and no buboes were noted. Blood and spleen showed *P. pestis* in large numbers.

The unruly soldiers of Hailar might be compared to the wild country folk of Fengchen (Shansi) who surrounded the railway-car occupied by the foreign doctors and nearly murdered them (Shansi Plague 1917). The two Russian railway doctors Dr. Churigan (in charge of Railway Hosp.) and Dr. Adolph (ambulance), as well as their two bacteriologists Drs. Petin and Askanoff gave our staff considerable help. One Russian dresser employed in the P.M. room was infected while dissecting and died three days afterwards. The Chinese Police Chief Lang was a young, active, modern trained man, without whose cooperation it would have been most difficult to successfully limit the spread. That he was attacked while on duty by the undisciplined soldiers proved his devotion to the cause.

2. *Dalainor.*

To understand the extreme infectiousness of pneumonic plague, the example of Dalainor offers an instructive example. Here is a coal mine owned by the Chinese Eastern Railway

and leased to a Russian Jew. The whole mine is scattered over an area about 3 miles square on flattish land, bounded on one side by low hills. There are five shafts worked by machinery to a depth of 150-200 feet in addition to shallow pits. The coal extracted is lighter and less lustrous than ordinary bituminous coal. It is in large slumps or slabs and does not ignite well, giving comparatively little heat and leaving an ash similar to that of wood. The daily output on a full day is 800,000 pounds. Except in the adjoining village, where a Chinese magistrate officiates, the administration is in the hands of the Russian lessee. The employees number 1000-2000 Russians and 4000 Chinese. The former occupy the higher positions while the latter form the bulk of actual miners. Besides these, there are 2000 dependents in the village. The Russians are housed comfortably in little cottages or open sunlit rooms built round the office building and have a club fitted with a small stage and restaurant.

The Chinese live mostly in semi-underground barracks scattered over the mining area. Each barrack measures 20 by 60 feet with accommodation for 60-80 men. The walls are built with coal-dust bricks, and the floors of mud. From the entrance, some ten steps lead to an underground five foot passage, on either side of which is situated a long *kang*-brick-and mud hollow structure raised two feet from the floor and heated in winter by a flue within. Four feet above this is a wooden-tier running the whole length of the room, used for the same purpose of sitting, sleeping and eating. In one corner of the passage near the entrance, is a small open brick stove where kettles may be placed for heating water. Beyond one or two slits on the roof fitted with glass for the admission of light, these underground dwellings have no sun or ventilation. They keep warm in winter at a minimum expense of fuel, but are stuffy and serve as terrible plague traps whenever an epidemic breaks out. The men usually receive twice the wages prevalent in Harbin. In the adjoining village, brothels, gambling dens, opium resorts and tea houses abound, and as may be expected such a population is not amenable to discipline.

Into this community there arrived from Hailar on January 2nd at 6 a.m. the sick friend of a mining coolie, who was among 17 living in a house. The sick man died at 6 o'clock the same evening. On the 7th, one of these 17 coolies was reported dead, on the 8th—one, on the 9th—two, on the 10th—eight, on the 13th—two, and on the 14th—the remaining three. Not one person in that unfortunate crowd survived. On Jan. 4th, in another barrack housing 30 men, one man got sick

and died. On Jan. 6th, in a third barrack containing 160 men another patient was found sick, sent to hospital and died a few hours afterwards. Of the contacts, over 42 were known to have taken the infection. On Jan. 11th, in a fourth but smaller barrack near the first one, four out of five inmates took ill at once, spat blood and died. One man remained healthy. On Jan. 16th one hospital attendant was reported sick, and the next day two more attendants sickened and died. Later on it was ascertained that 61 out of 64 coolies in one barrack died in 3 days as a result of infection from two outsiders. All this time, the medical control was in the hands of a Russian doctor Jejukavitch employed by the mines. The mine manager, Wedenikoff, an active man who had lived in America, appealed to us for help, and we immediately sent Drs. Chun and Yuan, and later on Drs. Hsieh, Chang and Chao, with sufficient dressers and equipment. I gave instructions that the contacts must not be kept in the infected barracks but removed to box cars in groups of 6-8, that they should stay out in the sun in the daytime, and that more police be lent to help in house-to-house inspection and to guard the hospital and cars. The rapidity of infection at Dalainor was principally due to the following causes:—

- a. Crowded barracks, especially when the men refused to report when sick. By the time a corpse was found, several others had already been infected and some escaped to spread the plague.
- b. Escape of sick and contacts from their places of confinement. Very often they returned again and again to the infected houses. Even in our Harbin Hospital we have seen one delirious patient trying to scale the wall and escape.
- c. The ignorance and lawlessness of the miners in opposing all anti-plague measures.
- d. The mistake made by the Russians in the early part of the epidemic in *shutting* up all contacts inside a barrack once a sick man was found, and only opening next day to sort out any further cases. A more crude way of isolation could not be imagined, but evidently this was done because of lack of spare buildings. (It was because of this that after the epidemic I insisted upon a Chinese doctor being associated with a Russian in the mines and the establishment of suitable blocks for isolation purposes).

The mortality quickly rose. In the third week of January over 30 deaths were deported daily, and in the fourth week

nearly 40. By Feb. 10th the mortality had registered 491. The highest point was reached on February 6th when 80 were recorded. When I visited the mines on February 11th all work had stopped, the dead were everywhere, and the population was well scared and more amenable to reason. In one house, eleven corpses were dragged out, but the friends still insisted upon going in to look over the papers left by the dead. The sanitary attendants while attending to the removal of the sick were often assailed, but fortunately the police was efficient and serious encounters did not take place. Under these conditions, the work of Dr. E. T. Hsieh who arrived on January 21st and his assistants (Drs. Chang, Chao, etc.) deserves every praise. Soon after arrival he fitted up a disinfecting and bath wagon, dressing room, with shower bath, and provided new clothes for patients and contacts. Contaminated clothes were disinfected in a special steam car. Assisting him were one ying of soldiers (90), 98 policemen and a staff of doctors, 11 dressers and 8 disinfecting assistants. The temperature in February was -20 to -25 C. 25 wagons were used for isolation. Steam from a small locomotive was used for disinfecting railway cars. The dead were cremated in an old brick-kiln. As coal was free, large quantities were used. At one time when the epidemic seemed very serious, it was thought that the whole mining population of Dalainor might be induced to live in the open air for a full week, warming to be effected by large coal fires, while the infected quarters were being disinfected. This open-air existence would also prevent further infections, and the epidemic would automatically subside. But the cold was too severe and not many dared to brave the ordeal. However, this method of prophylaxis should be undertaken in all tropical or subtropical countries, where the weather conditions are favourable.

The epidemic commenced to decline from February 10th, but fresh cases arrived from Manchouli. The last plague death at Dalainor was reported on May 19th, the total number registered having been 1017 (all Chinese except 4 Russians and 1 Japanese) out of a population of about 6,000. *Per capita* of population, Dalainor was the hardest hit of all places during the 1920-21 epidemic. In the outbreak of ten years ago, the mortality was insignificant, being 120 out of a population of 4000. On the staff, 8 soldiers, 1 Russian and two Chinese sanitary attendants died of plague.

3. *Manchouli.*

Manchouli or Manchuria Station is the western boundary town of Manchuria. Here ends the Chinese Eastern Railway, and the Trans-Siberian line begins. Mongolia is close by. The

population numbers 21,000, of whom nearly half are Chinese, the rest being mostly Russians. The recent political troubles in Russia have driven thousands of families into China by way of Manchouli, and the most destitute Russians may be found in this town. The plague epidemic of 1910-11 entered China from Dauria (Siberia) by way of Manchouli and claimed 400 victims locally. The outbreak of 1920-21 also started in Dauria and Abagatui and the first Manchurian infections were reported at Hailar. On this occasion, owing to the presence of thousands of poor Russians in Manchouli, who often lived underground with Chinese, the number of deaths among Russians was one-third of the total (334 out of 1141). In 1910, the Russian authorities took a leading part in the anti-plague work in this city; in 1920-21, owing to their disorganisation and lack of funds, our Service undertook the responsible share.

The first case was reported on Jan. 12, though a month previously rumours had been spread of the occurrence of plague in this city. On the 17th two more deaths occurred, and on the 21st, one. The total for January was 36. In February it had reached 475, in March it decreased to 318 and in April to 164. The highest point was reached on February 20th, with a figure of 70 (including 25 sick and 15 corpses found in one house). On Jan. 31st, 8 sick were found among 38 persons in a small inn, where one man had died of plague nine days previously. As at Dalainor in the early stages of the epidemic, no accommodation was available for the contacts who were either not isolated at all or were confined in the house where the sick had been. Hence the enormous number of sick and dead occasionally found in one building. After 40 wagons had been obtained from the Railway, matters improved considerably, but the multiplication of interests present was a great hindrance to efficiency. The mortality reached zero point on April 22 and stayed so for five days, but fresh infections were discovered on April 27, leading to a second small rise which continued until the 22nd of May. Dr. Kastorsky, a local Russian physician employed by the Municipality, gave the following reasons for the slow improvement:—improper organisation, no wagons at first for housing contacts, cold weather, fresh infections from Dalainor, Siberia and Mongolia, failure to report cases by Chinese, shortness of equipment, and the morphine habits of the Russian doctor in charge. The gentleman in question was dismissed in May.

The Russians had five doctors, six nurses, and 15 sanitary attendants on their staff. Our Plague Service personnel consisted of three doctors and four nurses among others. On the Russian side, there died of plague three nurses (including one woman), 15 sanitary attendants and one police. We lost none.

The Russian sanitary staff was mostly untrained, very careless about wearing masks in the presence of sick and dead. They smoked cigarettes while handling the dead, drank more alcohol than was good for them, and many were struck down unnecessarily. It was a sad story.

4. *Tsitsihar*

Tsitsihar consists of two parts—the station town on the main Harbin-Manchouli line and the Chinese capital city (Pukuei) joined to the station by a light twelve-mile railway. The station town has a population of some 5,000 people, while Pukuei contained about 90,000. It lies about one-third the distance from Harbin on the Harbin-Manchouli Railway, being 253 miles from Harbin and 623 miles from Manchouli. The journey from Harbin usually takes 8 hours. At Tsitsihar Station are large railway workshops, half the population being Russian. Pukuei is the seat of the Governor, who is supreme in all affairs of the Province. The modern hospital there was built by me in 1911 with funds provided by the local Governor, but since the opening its connection with the Plague Prevention Service has been indirect only, the annual expenses being defrayed by that city. On urgent occasions, I was consulted, as in February when General Sun Lieh Chen wired for me to see him and suggest the most appropriate measures for controlling the plague then rather threatening in the capital. The locations of the plague and isolation hospitals were excellent, the patients being housed in the commodious quarters formerly allotted to German war prisoners. Dr. Hsieh Chiu Kuan, a graduate of Formosa Medical College, was in charge, assisted by a staff of able young men. The police was active and made regular house-to-house inspection. As was usual in capital cities, much obstruction came from the influential classes in defying preventive measures, and the epidemic was unduly prolonged and claimed more victims than necessary. The first case occurred in a petty shop assistant who escaped from Dalainor and lived in the house of a friend, who was infected and died on Jan. 18. The wife followed and then five children, making a total of seven. These cases were hidden from the authorities, and when discovered other infections had already taken place. The Governor reported 11 cases (4 males and 7 females) up to January 23rd. The mortality records were as follows:—

For January 124, for February 661, for March 899, for April 44; total 1728. The males numbered 1390 and the females 338. The number of females was considerable because of the presence of many families in this city. Several deaths also occurred in the station area, corpses being actually thrown

out of the passenger cars on two occasions. Some of the Chinese employees in the workshops also succumbed. At Tsitsihar station medical inspection of passengers was introduced from February 1st, but this did not prevent some persons in the early stage of incubation from taking the train and then falling ill in their homes after arrival.

5. *Harbin.*

As in the previous epidemic of 1910-11, Harbin was the headquarters of the anti-plague organisation, from which all instructions and reports were sent. For ten years this city had been growing in size and population until in 1921 the latter reached a total of 300,000, including 4,000 Japanese, 100,000 Russians, and 2000 other Westerners. Harbin is the central station for trains proceeding to Changchun and the south; to Manchouli, Russia, and the west; to Suifenhö (Pogranitchnaya), Vladivostock and East Siberia. It is also the starting place for all river steamers plying on the Sungari and Amur. It collects grain, beans, oil, timber, skins, furs, etc., from the neighbouring districts for export, and is the main distributing centre for imported goods destined for North Manchuria and Siberia. It owns nearly 50 flour mills, 20 oil mills, 10 spirit distilleries and many breweries. Its Chinese merchants, mostly from Shantung, are, next to the Cantonese, perhaps the most forward in new enterprises of every description. Its regular lines of fine many-storied buildings along the main streets of the Chinese city (formerly a mass of mudsheds) are a lesson even to Shanghai. In fact, the city has grown ahead of the sanitary requirements, for there are no water works, no proper sewage system, no systematic collection of garbage and house wastes.

The richer Russians use water closets, but their municipal engineers do not yet understand the principle of septic tanks, still practise the cesspool method, and cart away the dejecta in iron tanks often to be conveniently dumped onto unoccupied fields near by. The Chinese city health work is still in the hands of untrained police officers, who only seek expert advice when a serious epidemic like plague threatens. Fortunately, the plague Prevention Service, with its largest hospital and principal laboratory situated in Harbin, has established a wide reputation throughout China, and by its successful control of epidemic and other diseases in the past has won the confidence of officials and merchants alike. When plague broke out in 1921, the ignorant classes were only half convinced, but they showed no open hostility. The real opposition came from the unruly soldiers, but even the hearts of these men were touched when they saw the tenderness and fearlessness with which

Chinese doctors and nurses attended their friends in this most dangerous of all diseases.

As soon as the plague at Hailar showed signs of spreading I called on Dec. 19, 1921, a meeting of the Chinese community at the Hospital attended by leading officials and representatives of the city. It was decided to start a local Plague Prevention Committee, to prepare 20 railway wagons for purposes of isolation, to establish a special detention house for the Chinese city, and to wire the Governor of Kirin for \$10,000 initial expenses. On December 21st a meeting of Chinese and foreign residents was held at the Customs House "to help in devising preventive and other measures against a possible epidemic in Harbin and surroundings." An International Committee was forthwith organised with Mr. R. C. L. d'Anjou (Customs Commissioner) as Chairman. This committee included all the consuls, higher Chinese officials, heads of the Railway, members of the Technical Board, business men, in addition to Chinese and Russian medical officers. Besides receiving reports it rendered much useful service in obtaining the cooperation and assistance of those who might be unwilling when approached individually. This Committee held altogether 18 meetings between Dec. 21 and May 23, after which it was dissolved.

The first plague case in Harbin was recorded on Jan. 22, when an ex-miner died suddenly in a house near the hospital after having arrived from Dalainor the previous day. Among four contacts (mother, sister, sister-in-law and male friend) who were admitted into our Hospital, only the sister-in-law took the disease on 26th and died the next day. This patient had fever, fast pulse and tightness in chest the first day, developed cough, later on spat blood, and died in the evening. Her sputum and blood showed the plague bacilli in large numbers.

In a small village four miles from the Chinese city another miner from Dalainor died of plague on Jan. 26. Three men living in the same room with him also died, and in addition members of three neighbouring families, with the result that in this small village of 73 huts and 400 souls, eleven persons died of plague between Jan. 26 and Feb. 6. Fortunately, owing to the early precautions taken at Harbin, all cases found were at once admitted into hospital, while the contacts were received in box-cars, each holding the members of one household (average six). It was evident that a trying time lay in front of us, and the whole efforts of the Service were concentrated upon limiting the outbreak to the smallest possible proportion. The plague wards could accommodate 60 sick, while the suspect wards had 50 beds. It was found that plague patients rarely survived beyond the second day after admission, as they were killed off so quickly. Sixty wagons were run on to a convenient

siding easily approached from the New Town, Pristan or Fuchiatien—the three main divisions of Harbin. These wagons included a sanitary car, a kitchen car, a supply car; the rest were for accommodation of the many contacts. As seen from the table, we had at one time over 600 persons to look after.

Statistics of Contacts in Isolation Wagons

Month	No Admitted	Sent to Hosp.	Plague	Non-Plague	% Plague
February	547	47	34	13	6.2
March	911	118	92	26	10.1
April	485	57	47	10	9.7
May	78	5	5	0	6.4
Total 4 months....	2,021	327	178	49	8.1 (AV.)

The work was under the supervision of Dr. Chaplick, who had a Chinese colleague as well as several assistants and a full sanitary staff. This was one cheerful spot in our daily duties, for upon its efficiency depended the saving of the lives of contacts. Temperature was taken morning and evening. As soon as any one showed signs of fever, especially with fast pulse, he or she was at once isolated and if necessary sent to the suspect ward of the hospital. Sometimes the rise in temperature was only temporary, and the patient was returned to the wagon. More often, the fever was followed by cough, red sputum and death. The 24 hours interval, usually between commencement of fever and cough, was most important, for at this stage the infective droplets had not appeared, and patient could be removed from his friends without any fear of their becoming infected. Should any carelessness or oversight take place, and the cough appear, then the inmates of the same car would run a serious risk of catching infection. The fact that the percentage of plague cases in the isolation cars reached only 8 out of 2021 admissions during the whole four months' epidemic showed that the work of Chaplick and his staff deserved every commendation. At Dalainor, where the same discipline was not maintained, 144 out of 655 contacts (21.9%) died.

The Russian settlement, comprising a larger area of land but much less crowded population, had a separate sub-office where the routine work of house inspection, diagnosis and disinfection was carried out, and the daily report sent to headquarters. All suspects and plague cases were sent to our Hospital in Fuchiatien, the few Russian sick (eight) being cared for in the Municipal Hospital. The Chinese city was divided into 5 districts, each with its corps of Police, dressers, inspectors, sanitary attendants, and disinfection squads. Patients were at once removed to hospital, while suspected corpses or dead found in the streets received spleen punctures. A simple method was employed:—Skin over spleen was painted

with Tr. Iodine, and a short sharp knife sterilised with iodine was inserted and some contents were withdrawn. The cultivations on agar were made, the knife being then stroked on two glass slides for microscopic examination. The whole operation usually took two minutes. In view of the very cold weather, strong wind and frequent exposure of corpse in the street, such quick operations were necessary. All cadavres found in the streets were sent to the common cremation pit to be burnt. Well-to-do and educated persons were sometimes thrown into the streets unclaimed, because the relatives after hiding them during sickness were afraid to report after death for fear of being isolated in the wagons. One of the most difficult problems of plague-prevention in China was this passive opposition of the populace in not reporting cases when alive and then throwing the bodies out when dead. If there had been co-operation between the public and the authorities at the beginning, the epidemic would have been more confined, but the cases were hidden and the families or friends were thus infected. This fear of isolation is prevalent in North and South China alike, and used to be the same in Japan until the new universal education of the masses produced its beneficial results. The dead in the plague-hospital, numbering 1312, were all cremated in two pits dug within its precincts. Each pit measured 12 by 12 by 10 feet deep. Large pieces of firewood were laid at the bottom, some bodies uncoffined were thrown in, then more wood, and so on. The fire was started by pouring two gallons of kerosene on the pile, and then lighting. The masses burnt fiercely in the open, because of the confined space, and even on cold windy days no difference was observed. In this way 80 to 100 bodies could be economically cremated every day with slight attention from two attendants. The fatty constituents of the cadavres helped to keep up the fire once it was lit, and at the end of the day only white crumbled bones were left as residue. Out of 1461 admissions into Harbin Hospital, 1312 were true plague cases, all died and were cremated. The remaining were non-plague and comprised such diseases as pneumonia, typhus, typhoid fever, tuberculosis with haemoptysis, influenza, bronchitis, catarrhal fever, etc.

Because of the large percentage of deaths, it was not surprising that rumours began to spread that we *took in* patients but did *not let them out*, and something uncanny must therefore have happened within the hospital compounds. Stories were current that our staff poisoned wells, flour and food in order to obtain reward of \$3 for each dead. My bulletin of March 13th contained the following:—

The past week has been a very anxious one for our anti-plague staff for the concentrated suspicion of and

prejudice against our policy of removal of the sick to hospital, isolation of contacts, systematic inspection of inns and other sources of infection, closing of theatres, low brothels, etc., coupled with the restriction of railway traffic and our inability to cure the plague victims resulted in numerous rumours to discredit our dangerous and humane mission and in some instances to actual threats of physical violence to our members. For instance, the Chief Medical Officer was accused of shooting the sick in the plague compound and was threatened with a similar fate should an opportunity offer itself, our house-to-house inspection doctors were on several occasions faced with revolvers and knives in the course of their duty, while the sanitary assistants were almost obliged to swallow some of the disinfectants used in the disinfected houses.

At Asheho a mob of sixty visited the isolation station, set free the two contacts confined there and chased the doctor in charge. It speaks well for the discipline of our staff that in the presence of so much provocation and the loss by plague of one of their leaders they have stuck to their duty and not resigned *en masse*.

To counteract these evil influences, we issued thousands of circulars, published a daily newspaper containing particulars about the sick and dead, reports from other infected localities, articles dealing with plague and health matters, etc. Our assistants also gave public lectures whenever possible and answered any questions that might be asked them by their audience. In fact, our staff were faced with open attack from resentful and frightened persons as well as unseen danger from the plague germ. But all stuck to their duties with a cheerful heart. The masses refused to take sanitary precautions and yet expected to escape infection. But the time came when our efforts were bound to succeed, the mortality curve showed a steady decline from April 10th and finally reached zero point on May 15th. The Harbin epidemic thus lasted nearly four months and claimed 3,125 deaths out of a population of 300,000, as compared with over 7,000 out of a population of 70,000 in the previous outbreak (1910-11). The death list might have been 30,000 or ten times greater if energetic measures had not been taken from the beginning. As it was, the epidemic was prevented from seriously invading the populous towns of South Manchuria and North China, less than 400 dying from plague in these latter regions as compared with 35,000 in 1910-11.

The daily ration of each contact kept in the isolation wagon consisted of:—meat $\frac{1}{2}$ lb; potatoes $\frac{1}{2}$ lb; carrots $\frac{1}{4}$ lb; white and black bread 2 lb; rice $\frac{1}{4}$ lb; sugar 4 lumps; tea twice

daily. As the rice, though expensive, was not filling enough for the northerners, Chinese bread (*mantou*) was afterwards substituted.

The Railway area employed 11 physicians (mostly Russians), 40 dressers, 60 special Police and 125 attendants. Our Service deputed for special duty in Fuchiatien 8 doctors, 14 dressers, 50 special police, 20 hospital attendants and 42 sanitary attendants. But of these there died of plague:—one Russian doctor (Sinitzin), one Chinese doctor (Yuan Teh Mao), one Russian dresser, one Chinese dresser, five hospital attendants in the plague compound, and one police sergeant. Seven burial coolies out of 18 employed in the public cemetery died of plague.

The River Sungari was opened for navigation on April 15 after all arrangements had been made with the Customs for medical inspection of passengers before purchase of tickets. Each steamer was obliged to carry a ship's surgeon on board. One suspected case of plague died on the first ship which left Harbin on April 16, but no further accidents occurred, and the river traffic was practically not interrupted.

A few interesting, sometimes humorous episodes may now be related in connection with our anti-plague experience:—

- a. Some of the most noisy detractors of our work were the native quacks who persisted in beguiling the public as to their ability to cure the disease. When patients came with fever and cough, they always gave hopeful prescriptions. If the sickness was not infectious, the patient got well and the quack's reputation jumped skywards; should it turn out to be true plague, both patient and quack often died. In Harbin alone, we recorded seven deaths among the old-style practitioners; one was the 'vice-president of the medical research society' whose body was thrown out into the street by his wife in order to escape isolation of the family. At Dalainor one quack after catching the disease passed it on to his wife and all four children. None survived.
- b. As it was easy for any one who could read a few ancient books on medical practice to hang out a sign-board, there was no lack of practitioners, and one influential group managed to persuade the Taoyin to hand over \$4,000 of government money for the establishment of a plague house where native treatment could be undertaken. This house was opened on April 1st, (Fool's day), with twelve native physicians on the consulting staff each drawing a salary of \$100. At their request we sent 10 certified plague cases to them

between April 1 and 4, but all died. On the 5th a letter was received from these worthies begging us not to send any more patients, as they had discovered that plague was incurable! The \$4,000 had been absorbed.

- c. The Inspection parties sometimes made mistakes. One man was sent in from the city because he had a fast pulse and had spat up something red. At hospital we learnt that he had been eating crab apples and the red spit was due to them. Needless to say, the man went home delighted.
- d. Most coolies were credulous and intensely ignorant about matters hygienic, but Dr. Chun (our Senior Med. Officer) got into conversation on April 9 with a Chinese carriage driver on his way home. The driver was young and pleasant and informed the doctor that on the previous day he was driving a fare when a policeman suddenly stopped his carriage and demanded where he was driving a *corpse* to. His live fare of fifteen minutes before had died of plague while in the carriage! The driver also said that he did not believe the rumours about doctors poisoning wells, food, etc. for he knew from experience that they were kind-hearted and treated all the poor well.
- e. Ten years ago the plague reached its zenith at the end of January. Chinese New Year happened to fall on the 31st and firecrackers burnt on this occasion seemed to have a marvellous effect in ridding the city of pest. In 1921 the plague *began* in January, and no amount of firecrackers on Chinese New Year seemed to stay its virulence.
- f. Many Chinese firmly believed in the efficacy of opium in keeping off plague, so much so that non-smokers were induced to try it during the epidemic. At Dalainor, a Japanese woman openly maintained an opium smoking and morphine establishment. Her business was most prosperous at the height of the epidemic. One day three plague corpses were found in her opium den. That served as an excellent reason for closing altogether her nefarious business, which at ordinary times might have required the cooperation of her consul.

Besides routine work, our laboratory in Harbin produced 60,000 cotton-and-gauze masks and 8,000 doses of anti-plague vaccine which were distributed throughout Manchuria and Siberia.

6. *Changchun.*

Changchun is an important grain centre, the southern terminus of the Chinese Eastern Railway (Russian-managed) and the northern point of the South Manchuria Railway (Japanese-managed). It is also the starting point of the Kirin-Changchun Railway (77 miles). It has a population of nearly 100,000 and is the seat of the Taoyin serving under the Governor of Kirin Province. In 1911 Changchun was one of the worst infected towns and lost more than 5,000 persons from plague. In those days it became also a great distributing centre of the epidemic, and the majority of infections in the southern localities could be traced directly to this source. When cases occurred in Harbin in January 1921, it was therefore most essential to keep Changchun as free as possible. Entire prohibition of railway traffic being impossible, the third class passengers were limited to a certain number each day. Hence, the limit of 50 for each train was fixed, the three daily trains thus conveying besides 1st and 2nd 150 third class passengers from Harbin to Changchun after undergoing medical inspection at the former station. Only persons with medical certificates could buy tickets. Medical officers also boarded the train at big stations and examined would-be travellers at intervening places. When the trains arrived at the Kwangchengtsu station (Erh Tao Kao), anti-plague officers examined all passengers, and removed those in the third class cars to special observation depots where they were quarantined for five days, after which they were liberated if found healthy. For this purpose, the Chinese authorities had prepared ten different inns and houses with large courtyards and the Japanese two places (used by them in 1919 for cholera cases), each accommodating about 150 persons. Railway cars were also utilised when accommodation was insufficient. Although leakages happened along the 150-mile journey, especially at Misatzu station (20 miles north of Changchun) where the clever ones dropped off and completed the rest of the journey on foot or in carts, this system worked fairly well, and only 77 cases of plague were recorded in Changchun during the whole epidemic. Here as elsewhere, some of the military hindered our work by unlocking the railway cars and allowing the passengers to escape before the quarantine station was reached. The Taoyin distributed money among these worthies and implored them not to break our rules. Our work at Changchun was rendered much simpler by the cooperation of the Japanese authorities, with whom we had our first meeting on February 24 and fixed the quarantine arrangements. The first Chinese doctor sent to organise the sanitary arrangements was Dr. Yu Shu Fen (a former student of Kitasato Institute, Tokio), who afterwards died of plague at Sangyuan, Shantung (March 24th). The local plague hospital

was situated near the Chinese Railway station (Erh Tao Kao). The commandeered inns were found ample for our quarantine purposes. On March 14th a fire occurred in one of the S.M.R. cars used for detaining two plague patients, one of whom had died and the other was unconscious. It appeared that the clothes of the latter caught fire from the small iron stove as he dropped down. The woodwork of the car was completely burnt. After Dr. Yu left, other physicians were sent by the Ministry of Interior to take charge of the work at Changchun.

7. *Suifenho.*

Suifenho or Pogranitchnaya lies on the eastern extremity of Manchuria, where the Chinese Eastern Railway leaves Chinese territory and enters Eastern Siberia. The town is situated on a beautiful plain surrounded by high hills. Chinese and Russian Customs officiate here. When plague from Harbin threatened to invade the eastern regions, I proceeded to this town on Feb. 16th and helped to organise the local International Anti-plague Committee on the lines of the Harbin one. Our Drs. Ling Ting Fan and Hsuan were placed in charge, assisted by the Railway doctor Gilleson. All trains arriving and leaving were inspected medically. The first plague case occurred in a Russian who died on 14th March. He had arrived a few days previously from Nikolsk in the Primorsk region (East Siberia). We ascertained that cases had already occurred at Nikolsk since March 8-10. A Russian Medical Officer from Nikolsk visited our station on Feb. 21, and later on wrote that he had seen cases among Chinese in his town on March 8th. The second Suifenho case was reported on March 16. Between this date and April 17th twelve cases were reported, ten among Chinese and two among Russians. Nine of the former were found in local inns, the tenth being caught at the station while trying to escape inspection. The first Russian patient has been described; the second was a nurse employed at the railway hospital.

8. *Other Infected Foci in Heilungkiang.*

Heilungkiang Province is separated from Kirin Province by the Sungari River. The Chinese Eastern Railway traverses this province from Manchouli until just north of Harbin, when it crosses the fine iron bridge and then enters Harbin, and thus Kirin Province. Practically all the important stations lying on this route were more or less infected by the plague. These included (counting from west to east) Dalainor (27), Hailar (175), Mentuho (282), Khingan (347), Buketu (371), Chalantun (487), Tsitsihar (623), Lamayantzu (707), Anda (757), Mankou (817), Tuichingsan (847), Station 62 (867), the figures after each city denoting the distance in miles from Manchouli. The

scenery for 200 miles between Mentuho and Chalantun was most beautiful, the railway making many picturesque windings. But plague spared no spot, however beautiful, and during the epidemic *Mentuho* reported 4 cases, *Khingan*-3, *Buketu*-25 and *Chalantun*—1. Buketu had large railway workshops employing 1500 persons, and among the deaths were six Chinese in one family.

At Anda 29 wagons were arranged on special sidings in the form of the letter V. Sixteen of these were on one side and comprised cars for plague, suspects, kitchen, disinfection, bath and *personnel*, while the other siding was reserved for isolation cars. The first case at Anda was found on March 19, and altogether 25 died. At the next station Tuichingsan, the soldiers interfered with the work, and 8 out of the 28 deaths happened among them. An important city *Hulan* lies opposite Harbin. In summer, there is river communication between these two grain marts, but in winter the frozen river is crossed by carriage, and hence the sick could escape from one to the other. The magistrate in charge was quite go-ahead and followed the instructions of the missionary doctor there, Dr. McKillop Young, as well his Chinese physician. Proper measures were adopted, and the total mortality from Feb. 7, when the first case was reported, to the termination in April was 322. In the 1910-11 outbreak, the deaths numbered over 4,000.

9. *Other Infected Localities in Kirin.*

From the second week of February, the plague began to extend its activities eastwards along the Railway. The first city to be attacked was *Asheho*, 40 miles from Harbin. A petty merchant Sun, whose people had died in Harbin, reached Asheho on Feb. 13 and stayed in the house of Liu. Sun spat blood on the 16 and died soon after. On the 19th Mrs. Liu (aged 45) died of plague. No report was made to the authorities. Then came the police, who removed Liu, his son and daughter to isolation. All kept well and were released. Unfortunately, Liu was again exposed to infection, coughed up blood on Feb. 27, and died the following day. The local soldiers encouraged a mob of 60 to invade the isolation hospital, set free two contacts, and actually forced the latter into the quarters provided for our dressers. This was too much for our Medical Officer, Liu, who left the town before instructions reached him from Harbin. The local magistrate was slow and did little to help the sanitary staff. The gentry undertook most of the responsibility and employed a pharmacist to look after affairs. In the workhouse, 40 inmates took the infection and died. In many cases whole families were wiped out. Deaths totalled 123.

Other important towns lying on the railway east of Harbin are:—*Imiampo* (153), *Shitao Hotzu* (210), *Hantao Hotzu* (255), *Hailin* (313), *Mutanchiang* (332), *Mulin* (402), *Machiaoha* (433), and *Suifenho* (512), the figures denoting distance in miles from Harbin. All these places were more or less affected, but in no case seriously because of the scarcity of population.

The tract going southwards from Harbin passes the following towns:—*Shwangcheng* (31), *Taolaichao* (76), *Yaomen* (101), *Misatzu* (125), the figures denoting mileage from Harbin. The magistrate at Shwangcheng was old-fashioned and allowed the plague to run its course. Fortunately, owing to the strict measures adopted at Harbin, only a few dripped into this city, and the total deaths numbered 134. The mortality in the other cities was not great.

From Changchun, the Kirin-Changchun Railway runs for 77 miles to the capital city Kirin, a residential centre. No cases were reported in Kirin, but a few were encountered along the line, having travelled by the road from Misatzu where they escaped quarantine inspection.

A large city *Yu Shu*, lying 100 miles south of Harbin but not on the railway line received its first infection on March 8th. The magistrate Mo had served as Chief of Police in Fuchiatien in 1910-11, and was therefore *au fait* with the elementary principles of plague prevention. He adopted the strictest precautions, sent his police to surround the city, telegraphed for some assistants from us, made house-to-house inspection, isolated contacts from the sick and cremated the dead. In this way he did good work. The plague claimed in his district 253 victims out of a scattered population of 50,000. Over one-third of these were females.

10. *Infected Localities in Fengtien Province.*

With our preventive measures adopted in the north and the watchfulness along the South Manchuria Railway, the only cases which escaped detection were those incubating the disease. At *Kungchuling* station (40 miles from Changchun) one sick passenger was caught on Jan. 31 in the train. He had travelled overnight from Harbin in the second class and had not therefore been quarantined. Three more cases were similarly discovered and removed.

a. *Mukden.* Mukden, being the capital city of Manchuria, the Governor-General was early communicated with. From the beginning he appointed me supreme chief of anti-plague operations, and wired to all subordinates including the military, to obey instructions. He had an efficient staff in Mukden, and was

assisted by Dr. Christie's staff and the Japanese. The first case occurred on March 29 at Siao Si Men (small west gate), found in an inn with 36 others. The servant at the inn caught the infection and died. A third case (corpse) was found on April 1 near the Chinese Railway station dressed in uniform. The fourth and last one occurred in the city, and strict measures prevented further spread. Hence the record of Mukden in 1921 was excellent. In 1911, about 5,000 persons died in this city.

b. *Koupangtzu*. An interesting localized outbreak was found at Koupangtzu district, where the Peking-Mukden line branches off to Yinkow (Newchwang) 90 miles away. A man Li arrived from Changchun on March 17 and lodged with a family named Wang at the village of Lo Chia Tun (9 miles north of Koupangtzu). A wedding was in progress and guests (male and female) were busy playing *machiang* (dominoes). Li died on the 19, followed by four of the Wangs and two others including the bride-to-be. Altogether seven died of plague. Kao Chia Tun (a village half a mile away) was infected by one of the guests, and lost 11 persons. The outbreak lasted from March 17 to April 4. All the sick had fever, cough and blood spitting. The local epidemic cost 18 lives and was due to one sick man arriving among a happy party.

11. *Vladivostock and Ussuri Districts.*

For some time reports had been received from Vladivostock as to the progress of the plague in that district. The first case was detected on April 9. From the first the Russian authorities appeared to have taken very strict measures. On May 24th Drs. Chaplick, Loshiloff and myself proceeded in a special car to Suifenho and Vladivostock. After staying the day at Suifenho, we arrived at *Grodoveko*, the Russian station on the other side. Here we met the Japanese military surgeon Iyeda, who a few weeks previously had come to Suifenho with twenty wagons for carrying out anti-plague work in Chinese territory. He informed me that the Russians closed up a whole house with the inmates as soon as a case of plague occurred. *Nikolsk*, where the Ussuri Railway proceeded northwards to Habarovsk (420 miles) was reached three hours afterwards. We found over 40 cars reserved for anti-plague work by the Russians. As soon as any suspected case was detected, all the other passengers in the same car went into isolation. The isolation cars were surrounded by barbed wire. The first true case of plague occurred on March 17 in a passenger probably from Harbin. A total of 16 deaths was reached in this city. Vladivostock (70 miles from Nikolsk and 488 miles from Harbin) is a beautiful seaport, the eastern terminus of the Trans-Siberian railroad. The Russian doctor in charge was Popoff, a keen man, former assistant of Zabolotny with whom I had worked in 1911. A revolution was then on,

and Popoff was on the point of being taken prisoner by the new masters (Kappelists). We found him at home, however, after three hours' search. His anti-plague organisation was somewhat complex. He employed nearly 1000 persons with a monthly budget of 200,000 yen. He had suboffices at Olga, Ugolnaya, Suchan and Habarovsk. At Vladivostock, there were 4 separate pest-houses, each with 8 beds (32), 14 isolation rooms each with 4 beds (56), and accommodation for 500 contacts in a military barrack. At the railway station were 4 wagons for isolation and one for sanitary staff. The staff consisted of 27 doctors at Y320 each, 40 dressers at Y230 each, 420 sanitars at Y140 each. Up to May 26, there had died at *Nikolsk* 16, at *Vladivostock* 240, at *Olga* 6, at *Ugolnaya* 9 and at *Suchan* 6. Among the staff there had died 2 dressers, 3 sanitars and 1 sister at plague hospital (out of 8 employed). Compensation in case of death was $\frac{2}{3}$ of monthly salary to widow or nearest relative for the rest of life. One dresser and one sanitar were infected while working in city; 1 dresser, 1 sister and 2 sanitars while in isolation hospital. Estimated population of Vladivostock (R. 100,000, C. 60,000, J. 30,000), Nikolsk (40,000) and Habarovsk (30,000).

The following are the mortality figures at Vladivostock:—

April 9	—	May 20	286	
May 21	—	May 25	81	
May 26	—	June 14	102	
June 15	—	Oct. 15	48	Total 517.

Two cases of bubonic plague were seen—one on July 10, the other on Aug. 10. Five infected rats were found on July 23 and two on Aug. 10. It would be interesting to know how widely the rats were infected. Dr. Popoff died the following October from poison by his political enemies.

12. *Infected Localities in Shantung and Chihli.*

a. Sangyuan District. About the middle of February, after the plague had been making some strides in Harbin, startling news came from south Chihli and the adjoining Sangyuan district of Shantung that many deaths from pneumonic plague had been detected in the villages. Medical assistance was rushed to the spot from Peking and Tientsin, including Chinese, American and French doctors. From Dr. C. W. Young's investigations, it appeared that a man Chang living at Chang Chia Wa village visited Tientsin about February 1 to buy a trousseau for his daughter, and died on the 4th, soon after his return. His whole family of 8 caught the infection and

died; altogether 40 persons fell victims in this village, every one traceable to Chang. Another man Wang visited the locality, returned to his home at Sangyuan on the Tientsin-Pukow Railway and infected his whole family, all of whom died except a small boy. Other families were also infected leading to about 120 deaths. The railway was practically stopped for some time, and only the express-trains to Shanghai ran. With later improvement, the service was extended, but the trains avoided the infected districts. The plague continued throughout April, and the last case was reported early in May. The usual train service was resumed on May 6th. Altogether 200 persons died of plague in these parts.

b. Chefoo and District. On May 4th, Chefoo was startled on hearing that the Russian Volunteer Fleet steamer *Kishinev* just arrived from Vladivostock, had two cases of plague on board. The Port Health Officer, Dr. Malcolm, who examined the passengers found that one patient died soon after the steamer left Vladivostock, and the second a short time before arrival at Chefoo. Nearly 200 deck passengers were on board. There being no quarantine station ready, the vessel was isolated with all on board. Five more deaths occurred the next two days, and by the 10th, the mortality had risen to 16, all from pneumonic plague. A deserted spot, Kentucky Island near Chefoo was found, where the survivors were landed, matsheds were hastily erected and food was brought ashore. 108 of the contacts escaped on the 5th, leaving only 4, and this produced a panic in the city. Fortunately, no further cases were reported. On June 8th the British steamer *Ralph Moller* reached Chefoo from Vladivostock, and reported three deaths from plague on the voyage. The passengers, numbering about 700, were removed to Kentucky Island, this time better fitted for the purpose. H. M. S. *Marazion* and two submarines stood on guard, search-lights were thrown on the island at night, thus preventing any one from escaping. The quarantine period having expired without mishap, the passengers were liberated. Strict measures were enforced in Chefoo city, but no local cases developed. In the meantime, five deaths had happened up to May 24 in a neighbouring village, *Ninghai*, ten miles east of Chefoo. These were traced to one man who escaped from the island on May 5th. There was no further accident, and the Chefoo outbreak closed with a mortality of 24.

c. Tsingtao. The British steamer *Kiangsi* from Vladivostock and Shanghai arrived at Tsingtao on June 4 with a case of pneumonic plague on board. This was followed on the 7th by the death of the mother from the same disease. No more cases were reported at Tsingtao.

D. APPROXIMATE EXPENDITURE OF ANTI-PLAGUE WORK

During this last epidemic, the expenditure was much less than that of 1910-11, when it ran into eight million dollars. So far as I can make out, the following sums were expended in 1920-21 at different places:

<i>Sum expended</i>	<i>Source</i>	<i>Authority</i>	<i>Locality spent</i>
Tls. 200,000	Canton Customs surplus.	Ministry Interior	Sangyuan district
Y. 170,000	S. Manch. Rly.	Japanese	Along S.M.R.
\$ 20,000	Mukden Governor	Governor	Fengtien
\$ 80,000	Customs	Ministry Interior	Changchun district
\$200,000	C. Eastern Rly.	Management	Along C.E.R.
\$100,000	Harbin Munic.	Municipality	Harbin district
\$ 50,000	Manchouli	"	Manchouli
\$ 40,000	Dalainor Mines	Management	Dalainor
Total about \$960,000.			

The Director of the North Manchurian Plague Prevention Service received \$150,000 from the Customs for operation in N. Manchuria. The Service itself spent only \$38,000 out of this sum.

Accounts are as follows:—

Received from Customs	\$150,000.00	Paid out to Harbin Taoyin	\$60,000.00
" " Heilung Governor	1,000.00	" " Heilung Governor	20,000.00
" " Miscellaneous sources	518.50	" " Plague Prevention Service	37,717.85
		Balance left with Customs accountant	13,800.65
Total	\$151,518.50	Total	\$151,518.50

Grand total for whole epidemic \$1,111,518.50

E. CHINESE REFUGEES FROM URGА

Early in February 1921, we received urgent requests from the civil and military authorities to aid ten thousand destitute Chinese refugees who were returning to China after the sack of Urga by the mad Baron Ungern and his hordes. As the plague was then at its height at the principal stations through which these people would pass, it was most essential not only to provide suitable cars but also to make all sanitary arrangements so as to prevent any possible infection. There would be no quarantine, provided the refugees were confined in the cars under proper escort, when the trains stopped at the various stations. Our Medical Officers at Manchouli, Buketu, Hailar, Tsitsihar and Harbin were instructed to take every precaution and to affix a seal upon the right sleeve of each person for identification. About fifteen trains were used altogether, food being provided them by the Chinese government at different places. The Russian and Japanese railway authorities co-operated heartily in this rescue work, and although the evacuation took over one month to complete, no mishap occurred, and the refugees reached their several homes without introducing one case of plague.

The following figures were passed at Manchouli:—

March	22	1,278
„	29	1,174
„	30	1,548
April	3	1,728
„	8	1,328
May	8	79
„	15	1,517
„	19	1,267
Total			<hr/> 9,919

CLINICAL OBSERVATIONS UPON THE SECOND MANCHURIAN PLAGUE EPIDEMIC 1920-21

BY WU LIEN-TEH, J. W. H. CHUN, R. POLLITZER

CONTENTS

- I. Introduction, symptoms and signs of the disease.
- II. Note on the beginning and decline of the epidemic.
- III. Observations on infectivity, epidemiology and incubation period.
- IV. Notes on symptomatology and therapy.

I. Introduction, Symptoms and Signs of the Disease

Our knowledge of pneumonic plague is based upon a series of outbreaks which have occurred in recent years. The Black Death which ravaged the whole of Europe and a large part of Asia might have been plague or a virulent type of influenza such as that encountered in 1918. The famous plague of London lasting from the sixteenth to the middle of the seventeenth century was mainly of the bubonic type. Among the annual plague outbreaks happening in India since 1896, some purely localised pneumonic epidemics have been encountered among the general bubonic type. The most prominent of these occurred at Kashmir, North India, in the cold season of 1903-1904 when 1443 cases were reported with 20 recoveries (all bubonic) towards the end of the epidemic. Two small epidemics have also been seen in the Gold Coast of Africa in 1908 and 1917.*

It was not until the first Manchurian outbreak occurred in 1910-11 with its huge toll of deaths (60,000) that the world's attention was riveted upon this most fatal of all infectious diseases. This Pneumonic Plague was carefully studied in all its aspects by Chinese, American and Japanese physicians at the time. The Shansi epidemic of 1917-18—also purely pneumonic in character and killing 16,000 persons—furthered our know-

* Two British missionary physicians, Drs. Lawson and Mackenzie died suddenly in June 1922 at Foochow (South China), probably of Pneumonic Plague. The former had attended a Chinese patient some days previously and developed the disease soon after. The second doctor attended his colleague and was himself infected 4 days afterwards. Bubonic plague is endemic in the city of Foochow.

ledge of the infection. The clinical features of Pneumonic Plague are thus described by Wu Lien Teh¹:—

Aetiology. Both sexes are equally susceptible and no person can withstand the disease once the organism enters the respiratory tract. Children are just as susceptible as adults, as evidenced by the Shuang-chengpu cases (1911) and last Shansi epidemic (1918). The disease attacks particularly poor people because of their surroundings; but doctors, nurses, students, merchants, Europeans, Chinese, Indians, Japanese, etc., have all been attacked and succumbed.

Symptomatology. The incubation period is usually two to three days, but this may extend to five days. The onset of the disease is usually abrupt; prodromal symptoms are rare. The disease begins with chilly sensations, but a distinct rigor does not occur. There is headache, loss of appetite, increased pulse rate, and fever. Vomiting rarely occurs.

Progress of the case. From 24 to 36 hours after the onset, the temperature has usually risen to 103-104° F. (39.4-40° C.) and the pulse rate to 110-130 per minute. Cough and dyspnoea appear within 24 hours after the onset of symptoms. The cough is usually easy and not painful. The expectoration is at first scanty, but soon becomes more abundant. The sputum consists at first of mucus which shortly becomes blood-tinged; later it is much thinner, frothy and of a bright red colour; it now contains enormous numbers of plague bacilli in almost pure culture. The typical rusty sputum of croupous pneumonia is not observed. The conjunctivae become injected, and the tongue assumes a brown or brownish-red coat. The headache becomes worse, the expression is anxious and face frequently takes on a dusky hue. Labial herpes is not seen. The patient complains of pain and a restricted feeling in the chest, but this is not severe. Apart from the disturbance due to the dyspnoea and their anxiety for their condition, the patients appear to suffer little pain. In the later stages of the disease, the respirations become greatly increased and the dyspnoea is very marked, the patients gasping for breath several hours before death. Cyanosis is then common and ecchymotic spots appear in several parts of the skin, the two conditions producing a black hue and thus giving rise to the name 'Black Death.'

The signs of cardiac involvement are very marked in advanced cases, the pulse becoming more rapid, soft, and feeble, until finally it cannot be felt. Death results from cardiac paralysis and exhaustion. The patients sometimes succumb after slight physical exertion, such as sitting up in bed or being moved. The temperature may decline to below normal before

¹ "Practice of Medicine in the Tropics" published in 1922 by the Oxford University Press. Vo II pp. 1038-1046

death. Delirium or coma is often met with before death. The urine in the later stages may show the presence of albumin. The diazo and indican reactions have not been observed in the few cases examined.

The spleen is usually not palpable, and the lymphatic glands are not enlarged. Bloody diarrhoea is occasionally observed, when *B. pestis* is found in large numbers in the stools. Leucocytosis is usually not present.

Physical signs. The physical signs in the lungs are often slight, even in cases well advanced in the disease. On percussion, dulness is often absent, and the vocal fremitus and resonance unchanged. In a small proportion of cases, however, localised areas of dulness may be distinguished. On auscultation, râles are frequently not heard except before death. When present early in the disease they are usually of the fine variety. Numerous râles are heard late in the disease, due to the oedematous condition of the lungs. Feeble respiratory sounds or pure tubular respiration over small areas are commonly heard. Not infrequently a dry pleuritic rub is heard at the side of the chest.

The heart is usually dilated on the right side, but not greatly. The heart sounds are fast and become feeble towards the end. In the early stages the second pulmonary sound may be accentuated, but this character is soon lost.

Complications and Sequelae. Pulmonary plague always ends in death with general signs of septicaemia caused by the *B. pestis*. The progress is very rapid, death usually taking place in two to three days from the onset of symptoms.

Diagnosis. The diagnosis of pulmonary plague is usually clear from the bacteriological examination of the sputum in which the bacillus is found in enormous numbers and in almost pure culture. A rise in temperature and an increased pulse are sometimes the only early symptoms noticed, and before the characteristic sputum appears the diagnosis may be doubtful, especially at the commencement of an epidemic or in isolated cases. However, the blood should always be examined microscopically and culturally, as the case may be one of primary septicaemia: A bacteriological diagnosis is the only certain method for excluding pneumonic infection due to organisms other than *B. pestis*, though the absence of clear physical signs in the lungs ought to suggest plague infection. As has been said above, coarse sibilant sounds and râles are seldom heard in plague pneumonia.

The sputum in plague is not purulent but of a thin, frothy, pink, or red nature. The cough is generally not so troublesome as in croupous pneumonia. The duration of the disease

is usually less than two days, and many cases succumb within 16 hours from the onset of symptoms. Cases may last three, sometimes four days, but rarely more.

Differential Diagnosis. Pulmonary plague has to be differentiated from typhus exanthematicus, ordinary croupous or broncho-pneumonia, relapsing fever, and influenzal pneumonia. The extraordinary virulence of the recent pandemic of influenza (1918) in which such a large proportion of pneumonia complications occurred with symptoms and signs unusually like those of pulmonary plague, has suggested to some observers the possibility of the causative organism being an attenuated form of *B. pestis*. But whereas in pneumonic plague *B. pestis* exists always in enormous numbers in the sputum, in influenza pneumonia a large variety of organisms, with or without Pfeiffer's bacillus, are present, but never *B. pestis*.

Prognosis. The prognosis is wellnigh hopeless, as no authentic case of pulmonary plague in either of the two great epidemics has been known to survive.

Treatment. This is also not satisfactory, and the best that can be done for a patient is to allay his sufferings. As soon as a case is diagnosed, the patient should be forthwith isolated, preferably in a hospital built for the purpose, and the contacts kept under observation. In Manchuria and Shansi, owing to the primitive conditions of living and to the conservative ideas of the inhabitants, it was most difficult to adopt strict quarantine measures. In an epidemic, unless the local police authorities are placed under the control of the trained sanitary officers, leakages in the organisation are bound to occur and the infection spreads unnecessarily. Capitalistic interests, official dignity, and medical efficiency are often at loggerheads on such occasions. The only remedy appears to be education, not only of the poor but also of the educated classes, in matters of quarantine and sanitation. In the Shansi epidemic, Young allowed the contacts to remain isolated in their own compounds, while the plague patient was removed to hospital. Each individual was taught to use a gauze mask, and although in some cases whole families were wiped out one after the other, the people were kept calm and learnt elementary measures of precaution. Any harsher measure would have produced riots. In the Manchurian epidemic 1911, although much opposition was experienced at first, the people followed the advice of the sanitary officers, even to the extent of permitting 6,000 bodies to be cremated in Fuchiatien alone. As a result of the successful measures in this city other cities followed suit, and cremated their own dead even when no medical officer was in charge.

Prophylaxis.

General. Apart from personal care, the strictest attention should be devoted to the adoption of general sanitary measures. On removal of a plague patient, his bedding should be collected and sterilized, preferably by steam or, if this be unavailable, by exposure to sun or steeping in a solution of cresol or izal.

The room and furniture should be disinfected in the usual way. All cups and small articles likely to have been grossly contaminated with sputum should be boiled. In case of death, especially in winter when the ground may be frozen to a depth of seven feet, the bodies may be confined in coffins and sent to a prepared pit for cremation along with others. This method, wherever available, is the most economical and efficient, and prevents the possibility of the bodies being gnawed by rats and thus leading to a bubonic epidemic. The management of an epidemic of pulmonary plague depends much upon the locality and upon the nature of the inhabitants. Where the people are educated to these necessary measures, as in Suffolk (1918), the outbreak should be at once suppressed, but in distant provinces in China, where the masses are not yet ripe for preventive medicine, considerable opposition may be expected. Wherever possible, the full co-operation of the local authorities should be obtained by the sanitary officers in charge.

Personal. No vaccine or serum discovered has yet been successful in immunizing a person against plague pneumonia. Hence, those in attendance upon the suspected and sick, such as sanitary officers, hospital assistants, stretcher coolies, disinfection staff, are advised to wear a proper mask as recommended by the Mukden Conference. There is no need to apply any deodorant or antiseptic to this mask, as its protective properties are entirely mechanical."

II. Note on the Beginning and Decline of the Epidemic

So far as we can ascertain, pneumonic plague epidemics have arisen as the result of earlier bubonic infections, which develop lung symptoms. These cases cough up masses of plague bacilli in the sputum, and under suitable circumstances such as overcrowding, winter weather, proper humidity, and indiscriminate spitting, infect others by the droplet method. Isolated instances of pneumonic plague arising in a laboratory have been recorded ^{2,3}. Simpson observed on several occasions "the first in a series of pneumonic cases occurring in an infected locality, the first generally arising in a house in which a large number of rats have died." Two interesting examples of

² Poech, Ueber die Beulenpest in Bombay im Jahre 1897, Vol. I.
³ Mededeelingen van de Burg. Geneesk. Dienst in Nederlandsch-Indie 1916|V|I.

pneumonic plague occurring on board steamers were found in the case of (1) the British freight steamer *Friary*, which lost 8 lascars out of a crew of 21 in 1901, and (2) British mail steamer *Nagoya*, which had eight deaths out a crew of 195 in 1919. Although no bubonic cases appear to have been observed immediately before the 1910-11 outbreak, it is very probable that it originated in this manner inasmuch as in the years preceding the epidemic sporadic cases of bubonic plague alternated with pneumonic cases in the endemic centres of Transbaikalia and Kirghiz steppes.⁴

From the 1921 epidemic we have been able to establish a complete link in the chain of evidence so far as human cases are concerned. Bubonic infections occurred in Transbaikalia in the late summer of 1920. Besides some cases at Kailastu (70 versts west of Manchouli) four were reported in September from Abagatui (Mongolia, 8 versts from Manchouli) and three from Dauria (60 versts west of Manchouli). Two Cossack soldiers were infected by the Abagatui cases and died, the last on the 20th September. After this no more was heard until October when plague was reported at Hailar. Most of the Hailar cases were observed by us. They are tabulated in Tables I and II, and described under another heading of this article.

After the bubonic cases, a series of the septicemic variety were met, leading later on to the purely pneumonic ones. The slow rate of increase in the number of cases from October to end of November was characteristic, and this differed markedly from the rapid spread in other places as soon as the pneumonic type was fully developed.

What then is the reason for this change in the character of the epidemic? In India, as we all know, occasional instances of pneumonic plague were seen among a large majority of bubonic cases, but these rarely developed into an epidemic. In Siberia, Mongolia, Manchuria and Shansi, on the other hand, the early bubonic cases showed a tendency to assume the pneumonic form, and although extensive outbreaks were comparatively infrequent, (in Manchuria only two epidemics in ten years) the danger of a pneumonic outbreak succeeding the bubonic infections has always to be borne in mind.

Barber and Teague⁵ tried by experiments with *V. cholerae*, *Sarcina* and *B. prodigiosus* to elucidate this phenomenon.

In their opinion the bacilli of pneumonic plague sputum were enabled to persist longer in an atmosphere with a low water deficit, such as is met with in the crowded inns of Manchuria in the winter. Hence the longer these droplets

⁴ Wu Lien Teh, l. c., p. 1038.

⁵ Phil. Journal of Sc., Vol. VII, Sec. B. No. 3, p. 157.

persisted, the greater would be the danger to the inmates. The Manchurian climate with a temperature considerably below zero C. fulfilled these conditions, and was therefore largely responsible for the propagation of the disease which would not happen in a warm country.

While ready to admit a cold climate as an important factor in the spread of pneumonic plague, we are not sure that the propagation of the disease is entirely due to this water deficit. It is possible that the origin of the Manchurian attacks can be traced to plague among marmots and not to an epizootic among rats. In this connection, it may be profitable to remember a statement made by McCoy⁶ that "Pneumonic plague in man rarely occurs from rat infections and it is an interesting and possibly significant fact that in plague squirrels there is a very definite tendency to pulmonary localisation, a condition which never occurs in plague rats." At the same time, the peculiar way in which the Hailar cases developed in 1920 strongly suggests a rat-flea infection.

Before discussing the decline of the epidemic it is necessary to review the observations made on the virulence of the B. P. in pneumonic plague. At the time of the Mukden Conference, experiments comparing the virulence of bubonic strains with those recently isolated during the pneumonic epidemic were made, and led to the conclusion that the B. P. in pneumonic plague possesses a higher virulence than many of the bubonic strains tested. Strong observed further there was a tendency to marked stability in the virulence of the B. P. Others contended that a longer incubation period was noticed towards the end of the epidemic, but these observations were probably not reliable⁷ and experimental evidence showed the virulence at the end of the epidemic to be undiminished.

Thus when the question of a spontaneous decline of the epidemic was raised it was evident that this supposed phenomenon could not be traced to a lessened virulence of the B. P. All observers agreed⁸ that, even if there should exist a spontaneous decline of the epidemic, the preventive measures were of the utmost importance in restricting the number of cases during the prevalence of the epidemic. Most of those taking part in this discussion were even of opinion that the preventive measures alone were responsible for the termination and denied the possibility of a spontaneous decline. On the other hand, some stress was laid on the possible influence of meteorological factors upon the course of the epidemic. The Conference thus decided: "The chief factor in the decline of the epidemic has probably been the preventive measures which were enforced,

⁶ Am. Journal Hyg., Vol. I, No. 2.

⁷ Mukden Report, pp. 73-74.

⁸ Mukden Report, pp. 55-75.

either in accordance with scientific methods or by the crude efforts of the people to protect themselves. Climatic influences may have contributed indirectly, or even directly, toward bringing the epidemic to end, but the evidence presented on these points is inconclusive. The decline has not been due to any loss of virulence of the bacillus."

As shown in our paper on the postmortem findings of 1921 we found one factor which might possibly play an important role in the decline of the epidemic. This was the prevalence of pneumonic cases at the height of the epidemic as compared with their scarcity towards the end, at which period only pulmonary cases prevailed. These were far less infectious, because of the absence of sputum, than the strictly pneumonic. Being thus rendered less infectious, the "spontaneous decline" of the epidemic, even in foci unattended by sanitary organisations, might be easily explained.

III. Observations on Infectivity, Epidemiology and Incubation Period

In these epidemiological and clinical notes only such observations will be mentioned which seemed rare in former outbreaks or not mentioned at all.

Instances were seen where contacts had been certainly exposed to infection and yet remained healthy.⁹ Chun ("Salient points about the recent pneumonic plague in Harbin") mentioned the fact that many patients suffering from ordinary diseases were sent to the hospital as plague cases. It was impossible for the inspection squads to be always right in their prima vista diagnosis, especially with the Russian squads in the Railway area where they had great difficulty in ascertaining the history of the sick.

So, instead of taking the risk of leaving the possibly plague patients in their homes, they sent many "suspects" to the hospital where special wards were provided for them. On admission every precaution was taken by us to separate the real plague cases from the suspects, while patients, who suffered from ordinary diseases were sent home wherever possible.

In spite of the exercise of much care, it happened that patients suffering from ordinary diseases did sometimes stay in the plague wards during the 1921 epidemic, the same as in the 1911 outbreak.¹⁰

- a. A man, aged 26, was admitted into the plague ward, as he expectorated sanguinous sputum, and remained there eight days. During this period, three other plague patients were put in the same ward and died. Further investigation into his case revealed the fact

⁹ Mukden Report, p. 389.

¹⁰ Mukden Report, p. 295.

that he was really suffering from croupous pneumonia. Two animals were injected with his sputum and both died, showing only pneumococci in their organs. This patient made a good recovery afterwards.

- b. Three Chinese from Irkutsk were admitted into the plague ward. They were refugees and starved. They passed through the plague infected region on their way to Harbin and were thought to be suffering from plague. One of them did die of plague soon after admission, the second man died of inanition, but the third man, named An did not die after spending 11 days in the ward. This man was particularly interesting, because he volunteered to work as ward attendant, and after a few weeks' duty was infected with plague and died. So one could not consider him as naturally immune because he did not at first "catch plague during his stay as a patient in the ward.

In connection with first case (a) the possibility of the protection afforded by a pneumococcal infection against plague has to be considered.¹¹ We accidentally came across some apparent evidence to support this when injecting rabbits with plague cultures contaminated with *Diplococcus lanceolatus*. The rabbits died quickly, but at P.M. only pneumococci could be recovered. A very interesting instance was observed in Dalainor. On 6th February, three plague corpses were discovered under a bench in an opium den kept by a Japanese woman. The owner and sixteen men found smoking in the den were all removed to the isolation wagons, but none of them was subsequently attacked with plague.

Dr. Hsieh, in his report on these cases, remarked that the local people believed that opium smokers were immune to plague. Of course, there is no evidence that this belief is justified and so one may put it in the same category as the supposed prophylactic use of alcohol against cholera.

As a spoke in the wheel, we may mention that while conducting P.M. examinations, we noticed many plague corpses with morphine-injection marks.

As in the previous epidemic¹² we also observed instances where people attended on their sick relatives and yet did not get infected. A woman, age 24, looked after two plague stricken relatives and when they died, she attended on her father. Because she had fever (afterwards found to be due to an alveolar abscess) she and her father were admitted into the same room in the suspect ward. On the next day, her father died of plague pneumonia. She herself recovered from her

¹¹ Mukden Report, pp. 157-158.

¹² Mukden Report, p. 232.

abscess three days later and was sent home after a further period of 5 days.

We found that in several instances children escaped infection while the rest of their family succumbed to plague.

1. In Shwang Cheng Pu, 13 members of a family living in a compound died within two weeks, leaving a child one year old.
2. In Harbin, a boy of 11 remained well while six members of the same household died within two days.
- 3, 4. In the hospital we had two lots of children who did not get plague, though their mothers succumbed.

We can only cite these cases¹³ as curious and lucky escapes and cannot deduct therefrom evidence of immunity of children from plague.

Out of 3412 prostitutes in Fuchiatien, only 12 deaths from plague were reported. Twelve servants (men) also died. The brothels were closed for one month at the beginning of the epidemic but were reopened later on and some deaths among the guests were reported.

The theatres were also closed at the beginning, but some of them were reopened. Several cases of plague occurred among the stage people living on the premises.

Four instances were on record where a plague patient suffering from fever and cough was travelling in a crowded railway car on the Harbin-Changchun line (150 miles). On 2nd February, a dying man with haemoptysis was found on the train at Yaomen. The other 47 passengers were sent on to Harbin for observation, but as they remained healthy after six days, were liberated. On 16th, 20th Feb. and 15th March, 37, 47 and 30 passengers respectively travelled on each occasion with a sick man but no one developed plague subsequently. Jasienski reported a similar instance in the previous epidemic.¹⁴

In contrast to the above, we may cite a few interesting cases of infection by contact.

1. A prisoner (convicted for theft) was ill with plague after 2 days' confinement and was sent to the hospital where he died. Three days later 5 other prisoners from the same cell died of plague.

2. In an iron foundry, a man died of plague. To avoid inconvenience, the owner induced a military sergeant to rush the body to the burial ground without notifying the police. Soon after 2 persons including the owner died, one after the other. Three remaining contacts (including brother of owner) were sent to the isolation wagons.

3. In a Cold Storage Company, a fitter visited his father who was ill with plague in the city. After his father's death,

¹³ Mukden Report, p. 172.

¹⁴ Mukden Report, p. 220.

he got four friends who were living with the other labourers of the company to carry the corpse and deposit it on the street. The next day he was ill with plague and on the day after that his four friends were also plague stricken. They all died in the hospital 48 hours after admission. Meanwhile, other cases occurred in the labourers' quarters and the contacts ran away to different places. One of the fugitives infected a household of 12 persons.

Sanitary Squads and Burial-Ground Personnel

All available data from Harbin and Dalainor were tabulated in Tables III and IV.

Mortality among the Sanitary squads was high, especially in Dalainor where it reached 50%, the highest figure reported in the previous epidemic.¹⁵

When comparing this high mortality among the sanitary coolies with the clear record of our dressers, one must bear in mind the fact that the coolies were certainly more exposed to infection than the dressers. Moreover, the dressers were more aware of the danger and took care to follow the precautionary measures exactly.

To account for the high percentage of infection among the sanitary coolies and sanitary police, one more factor has to be considered, namely, the stealing of money and clothes belonging to the corpses. Thus two of the coolies and one policeman were known to have committed thefts of this nature in the plague wards at night-time. As they died soon afterwards of plague, we may say that some indirect evidence of the infectivity of the clothes and money is established.

This factor also deserves notice when we consider the high mortality of the burial-ground coolies in Harbin. In Dalainor, however, Dr. Hsieh did not find any plague deaths among the burial-ground coolies, although they undoubtedly committed thefts.

Incubation Period

The average incubation period was three days, varying from 2-6 days. Longer periods were also observed as in the previous epidemic. Thus:—

1. One man was sent from the Isolation wagons to the suspect ward because he had fever. He had continuous fever for 7 days, but repeated examinations of his non-sanguinous sputum proved negative, until the 7th day when he showed B. P. and blood in his sputum.
2. A case was reported from Asheho of a man dying 8 days after the death of his wife.

¹⁵ Mukden Report, p. 194.

3. A woman, aged 17, was sent to the Isolation wagon after the death of her husband. For 6 days she had a normal temperature. Before going home, she had a bath. She then felt ill and giddy but was able to walk home, a distance of half mile. Soon after she became very ill and was admitted into our hospital after six hours. On admission she had no cough or expectoration. But on the next morning plenty of blood and B. P. were found in her sputum and death occurred 24 hours later. P.M. examination (No. 6) revealed plague pneumonia.

IV. Notes on Symptomatology and Therapy

a. Non-infective period at beginning of disease.

We can say without hesitation that as a rule there is a non-infective period of about 24 hours during which the patient has fever and quick pulse but no cough or bloody expectoration. This is the time when he may be moved to hospital without danger to others.

As an illustration, Dr. Yuan Teh Mao, chief of the inspection squad, felt a little unwell on the morning of the 5th day after infection. He was on duty nevertheless, attended a meeting of 26 people in the afternoon and mixed with the other members of the staff for the whole day. In the evening, he went to bed with fever 120° F. Next morning, he had a little cough and B. P. and blood were found in his sputum. None of the 60 persons he came in contact with the previous day were infected.

Chung¹⁶ reported a similar instance (case of a Japanese doctor).

Zaboloty¹⁷ remarked on this non-infective period which he estimated at two days.

b. Observations upon cough and hæmoptysis.

The cough of the plague patient is at first dry, but very soon he expectorates a liquid, frothy and bright red sputum. Frequently, there is a considerable amount of bloody sputum, especially among young patients, so that the floor and bedding are covered with blood. For instance, a patient was able to spit into 12 Petri dishes within a few minutes and was still coughing and spitting afterwards. Sometimes profuse hæmorrhage was observed immediately before death.

Epistaxis was not observed. Bloody froth was often seen at the nostrils of patients *in extremis* and corpses.

Cases with no cough or sputum will be mentioned later on.

¹⁶ Mukden Report, p. 226.

¹⁷ Mukden Report, p. 91.

c. Pain.

There is not much pain in connection with plague pneumonia, in contrast to ordinary pleuro-pneumonia, though a few exceptions were observed. Nearing death, the patient is very short of breath, mildly delirious, often tries to sit up or struggles to go out into the open air. He is not conscious of much pain owing to CO² intoxication and incipient oedema of the brain. Some cases in Manchouli were reported to keep cheerful in spite of alarmingly profuse haemorrhagic expectoration.

Active delirium is rare. This may be due to the gentle and stoical nature of the patients who are mostly coolies and are not addicted to alcohol.

d. Symptoms of in-coordination.

The loss of co-ordination of the voluntary muscles of the legs together with other nervous symptoms render the plague patient unsteady in his gait. To the uninitiated, he resembles a drunken man and several drunkards have been sent into the hospital as possible plague cases.

This staggering gait has not been explained on anatomical grounds. Simpson¹⁸ ascribed this symptom "to a progressively intoxicating effect of the plague virus on the nervous system." Our records mention the following:—

1. A man, aged 24, on admission into the hospital, stumbled on to a long wooden stool and fell head first, almost turning a somersault. After lying on the ground for five minutes he got up with great effort and sat on the stool, still unable to hold his head up. He rested on another patient lying on the stool next to him.

He could not answer questions. His knee-jerks were not exaggerated and as far as one could judge, he had no nystagmus. He died half an hour afterwards with increasing dyspnoea and cyanosis. At P.M. (No. 16) the meninges were found to be congested while the brain was oedematous. The petrous portion of the temporal bone was removed for further examination.

2. Another man was closely observed. He could walk, though unsteadily, to the ward. After five minutes, he wished to come out, but was now able to walk only with assistance. He evidently felt giddy for he held on to the drain-pipe for three minutes. Then he fell down gasping and died after 8 minutes.

e. Clinical observations upon urine and faeces.

Only little attention was directed towards the urine and faeces of plague patients in the previous epidemic. Kasai examined six samples of urine. He found them clear or slightly cloudy, acid in reaction, and with a specific gravity of 1024-34. Albumen was found in 2 out of the 6. Diazo reaction, indican, sugar and blood tests were negative.

A systematic analysis of 30 urines was conducted by Chun, in 1921, who noticed that the chlorides were diminished in all cases, a feature usually found in ordinary pneumonia and bubonic plague^{19 20} and other fevers. The absence of blood is noteworthy. Other findings corresponded with those changes met with in acute febrile diseases. No case of *intestinal infection* during life was observed, in contrast to clinical reports of cases during the previous epidemic.²¹ At the P.M. (No. 12) of a young subject (age 10) liquid stool was seen flowing out of the rectum. The mesenteric glands were enlarged as usual. Nothing abnormal was detected in the alimentary canal. Cultures of B. P. were obtained from the ileum and colon as in other cases.

f. Duration of disease.

Tabulation showing duration of stay of plague patients in hospital:—

No. of Patients Recorded	Average Stay in Days	Longest Stay	Average Duration
February .. 85	2.0		
March 476	1.7	9 days	1.8 days
April 561	1.7		
May 6	2.1		

In the majority of cases, patients were sent into the hospital within a few hours of their illness. So the above average duration 1.8 days, may well be taken as the duration of the illness, and corresponds with those findings of Kasai²² and Strong.²³ It is 24 hours longer than what Haffkine²⁴ found in Harbin Jan. 1911, however. Emphasis must be laid on the fact that the average duration of illness towards the end of the epidemic (April and May) was no longer than at the beginning—another proof that the virulence of B. P. was in no wise diminished.

19 Simpson p. 292.

20 Jennings, Manual of Plague, p. 89.

21 Mukden Report, pp. 165-183.

22 Mudken Report, p. 177-178.

23 Mudken Report. p. 431.

24 Mudken Report, p. 106.

Kasai (l.c.) recorded a few instances where the patients survived 5—7 days. We were able to observe a case of nine days (see Table V). A man, aged 32, (P.M. No. 11) came to the hospital voluntarily on the 16th March. After admission, he had fever continuously, 102° F., feeble pulse and B. P. and blood in his sputum. Beside the abnormal length of illness, his sputum assumed a muco-purulent and rusty red appearance during the later stages, though plenty B. P. were present. On the 23rd March a flask of *B. subtilis* emulsion was injected subcutaneously with some apparent benefit. On the morning of the 24th, he was semi-conscious though he could be aroused. Towards noon, he became stertorous and died half an hour later of heart failure.

At P.M., broncho-pneumonia was found in four lobes; no other unusual features, except an anaemic infarct in spleen which is the only one found in our P. M. examinations.

g. Cases among children.

A few cases of plague among children and women came under our notice. Economic conditions force the coolies to migrate from Shantung and Chihli to Manchuria leaving their womenfolk and children behind, and to live in crowded lodgings. These factors account for the high percentage of coolies among our patients and the small percentage of women and children.

Both clinical and P. M. observations show that the children succumbed to plague more easily and quickly than adults. For, out of six P. M. examinations of children, only two had well marked pneumonic changes. One pulmonary case was seen at the height of the epidemic, two had slight pneumonic changes, and one had a single patch of pneumonia. The case of a still born foetus is fully described in the P. M. article.

h. Non-pneumonic cases in 1921 epidemic.

i. Bubonic. We have been the series of bubonic cases beginning with those at Abagatui (August), Kailastu and Dauria (September). One of the Abagatui cases was treated at Manchouli Hospital and recovered. Two Cossacks infected by this family developed buboes and died (the last on September 20th). Out of the possible thirteen, only one recovered.

Then came the Hailar cases, numbering at least eleven, the earlist ones, as in the previous thirteen, being entirely among Russians.

Throughout the epidemic, which now assumed a pneumonic character, we were on the lookout for further bubonic cases, but the following were the only ones recorded in this series:—

1—One doubtful bubo in a Russian man from the Priamur district treated at Suifenho. This patient died on 9th March.

II—One bubonic case reported in February by a Russian lady physician of Chita as occurring among 22 cases at six cities in Transbaikalia.

III—One bubo in right groin of a Russian woman Bulak, aged 25, pregnant four months. We saw this patient on April 5th and following days. Patient was married and lived in a small house in Pristan doing her own household work. In the same compound some Chinese were living, but no plague cases had so far been reported here. On March 31st she experienced a prick in right foot and noticed a small swelling in the groin next morning (Ap. 1st) with fever. When admitted on April 4th, both right femoral and right inguinal regions were swollen, each of size of walnut. Dr. Askanoff of the Railway Hospital punctured the femoral bubo and noticed a few definite *B. pestis* in the smears (15 B. P. on one slide). Cultivations and animal experiments with gpus. gave positive results for B. P. Bubonic plague was therefore diagnosed. On the day of our examination (Ap. 5th), there was considerable swelling, the inguinal bubo measuring size of a small apple. and the femoral slightly smaller. Both swellings were painful and tender. Ichthyol was applied to the region. 240 c.c. anti-pest serum (Japan) was injected on April 4th, and repeated next day. There was marked induration at site. Patient looked flushed but not very ill. Temperature undulated about 39.5 for some days. The swelling gradually disappeared with fall of temperature and patient eventually recovered. A full grown baby was born at proper time well.

IV—Russian man was admitted into Manchouli Hospital on May 26th with a marked right submaxillary bubo. He had no cough or sputum. He looked very ill and had high temperature. He died on May 29th after 4 days' illness.

V—Out of 496 cases of plague at Vladivostok all were pneumonic except one Russian with a right inguinal bubo, who was admitted on July 10th and died two days later. About this time systematic rat examination showed two positive infections on July 2nd, and again five between July 18-23.

VI—Murakami (Japanese Military surgeon) also reported a doubtful bubonic case in Vladivostok on August 10th.

Of those six cases—all in Russians, two were, doubtful, two were in right limb, one was in submaxillary region, while the location of the sixth (Transbaikal) one was not stated. The only case we have full data of was that of the Russian woman. who was seen in hospital 4 days after the alleged prick. If the swelling was due to this prick as she believed, it must have developed unusually quickly for an ordinary septic in-

fection. But a scratch due to fleas might easily have been mistaken for a prick. It is likely that the mode of entry of the B. P. in her case was not dissimilar to that of any other bubonic case, namely through a rat flea. The fact that no other case of plague—either pneumonic or bubonic—occurred in the same compound tended to support this theory of a stray infection.

Should any accidental sputum touch the abraded skin of a hand, the bubo would then appear in the axilla, which none of our cases showed. Still the mystery remains as to the appearance of the rare instances of bubonic plague among thousands of pneumonic cases seen. We have no explanation to offer as to their actual cause. It is noteworthy that no cases of bubonic plague developed towards the end of 1921 epidemic as in the case of Kashmir.

ii. Pulmonary cases. In two separate articles we have drawn attention to the occurrence of strictly pulmonary cases towards the end of epidemic. Cases apparently similar to these pulmonary ones were met with at postmortems **during** (not end of) the epidemic of 1910-11. Similar clinical observations were made at that time. Christie maintained that "septicemic" cases were comparatively frequent and formed about 10% of all cases; however, owing to the rapid death in this type of the disease very few patients died in the hospitals. Hill after examining the rough clinical records of 15,000 cases in 1911 said "the septicaemic cases without pneumonia constituted a very small proportion of the whole, probably about 1% or less."

Our own observations tended to confirm this rare occurrence of the pulmonary type at the height of the epidemic. It is true that at this time our autopsies were mostly performed on cases which had been observed clinically and not on cadavers picked up in the streets, but there is no evidence to prove that the street corpses had succumbed to the type described by Christie. According to our observations most of these cases had been sick and died inside the houses and were then thrown out in order to avoid isolation of the rest of the household. It is interesting to recall P.M. 17 (child of one year) found dead in the street at beginning of April, and showing not the pulmonary but only slight pneumonic changes. Should any further occasion arise, it might be wise for investigations to be directed upon these street corpses during the height of the epidemic.

In our records we found seven instances where patients died suddenly without apparent signs of pneumonia. One of

these may be mentioned here. A girl aet. 3 (No. 4 of our P.M. series) was admitted as contact on March 4th because her mother suffered from plague. Swab taken from the throat of the child showed pneumococci in smears and thin gramnegative bacilli; in cultures, micrococcus catarrhalis and a few bipolar stained gramnegative suspicious bacteria. The child was placed on March 4th. under the care of an elderly woman, who however took ill on March 5th. and died the same evening. The child was then placed under the care of two men convalescent from typhoid. She looked well and bright the whole day of March 5th and had no cough or bloody expectoration. She died early on March 6th. At P.M. slight reaction in larynx and trachea and slight pneumonic changes in lungs were found (Group B. of our cases). Thus this case cannot be considered as pulmonary in the strictest sense of the word, but belongs to the same category as Case 17 mentioned above. Similar cases were met with in our experience where patients died suddenly from apparent pneumonia and did not show cough with bloody expectoration until immediately before death.

i. Therapeutic measures.

No form of treatment was found of any avail. However, mention should be made of several attempts to save the lives of our plague patients. No effort was spared in trying to help Dr. Yuan, our able colleague, and others of our staff who were infected in the course of their duty.

The therapeutic agents used were:—

1. Antiplague serum (Japanese source) 40-80 c.c. (repeated several times), both intravenously and hypodermically.
2. Methylene blue **per os**.
3. Eusol 20-120 c.c. intravenously.
4. Formalin solution (I-200 strength) 100-200 c.c. intravenously.
5. Neoarsenobenzol 0.4-0.9 grm intravenously.
6. Sod. gynocardate 2 c.c. of 1% solution intravenously.
7. B. Subtilis emulsion 25 c.c.
8. B. H. emulsion 25 c.c.
9. Electrargol 10 c.c. hypodermically.

Some of the drugs were repeated on the same day, and were given as soon as the diagnosis was reasonably sure. As reported, they did not cure the patient, nor did they prolong life. On the other hand, such drugs like neoarsenobenzol, eusol and sod. gynocardate appeared to hasten death, as the patients generally died within 24 hours..

TABLE I

Bubonic Plague cases, observed at the beginning of the epidemic in Hailar.

No. Case	Residence	Date taken sick	Date admitted to hospital	Date death	Observations
1 Mrs. Tarelkin (Russian)	Ry. bridge barrack	22. Oct.	23. Oct.	Fever, Angina. swollen neck. rigor
2 Son of Mrs. T. (Russ. aet. 9)	—, —	Do.	Do.	Fever, swollen neck. (Was seen by Russ, hospital Felcher).
3 Soldier Chen (Chinese aet 24)	—, —	23. Oct.	25. Oct.	Fever swollen neck. rash on body.
4 Son of Mrs. T. (Russ. aet. 16)	—, —	15. Nov.	15. Nov.	17. Nov.	T40, rash on body, small ill defined buboes, Autopsy,
5 Son of Mrs. T. (Russ. aet. 18)	—, —	Do.	Do.	21. Nov.	T38. bubo left groin Autopsy.
6 Soldier Chang (Chinese aet. 19)	—, —	17. Nov.	19. Nov.	Bubo left armpit
7 Mr. Tarelkin (Russ. aet. 43)	—, —	18. Nov.	18. Nov.	Bubo left groin recovery (See Tbl. 2)
8 Soldier Chao (Chinese aet. 21)	—, —	21. Nov.	24. Nov.	25. Nov.	Fever, bubo left axilla.
9 Chinese coolie Li	Yi Shun Tien	?	?	13. Nov.	Spleen Punct. Septic. (?Bubo)
10 Wang	Yu Tai Chan	?	?	2. Dec.	Septic. (?Bubo)
11 Han	Soldier's inn	?	?	8. Dec.	Septic. (? bubo)

TABLE II

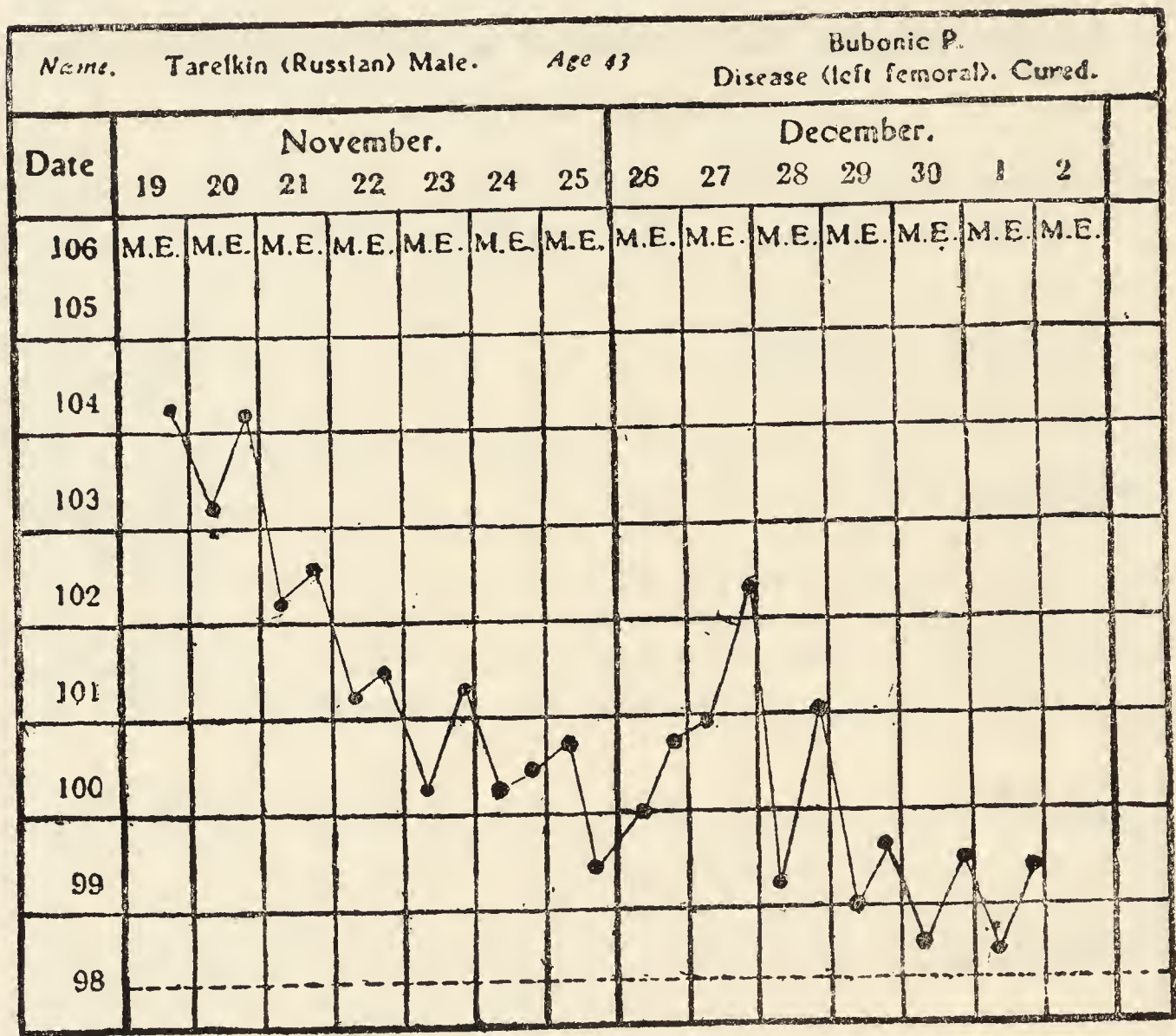


TABLE III

Tabulation of Sanitary personnel Harbin Hospital.

Character.	No. employed.	No. infected.	% infected.
Doctors	8	1	12,5
Dressers	20	0	0,0
Sanitary coolies ..	13	5	38,4
Sanitary police ...	10	1	10,0
Others (cook, etc.)	3	0	0,0
Burial coolies	18	7	38,8

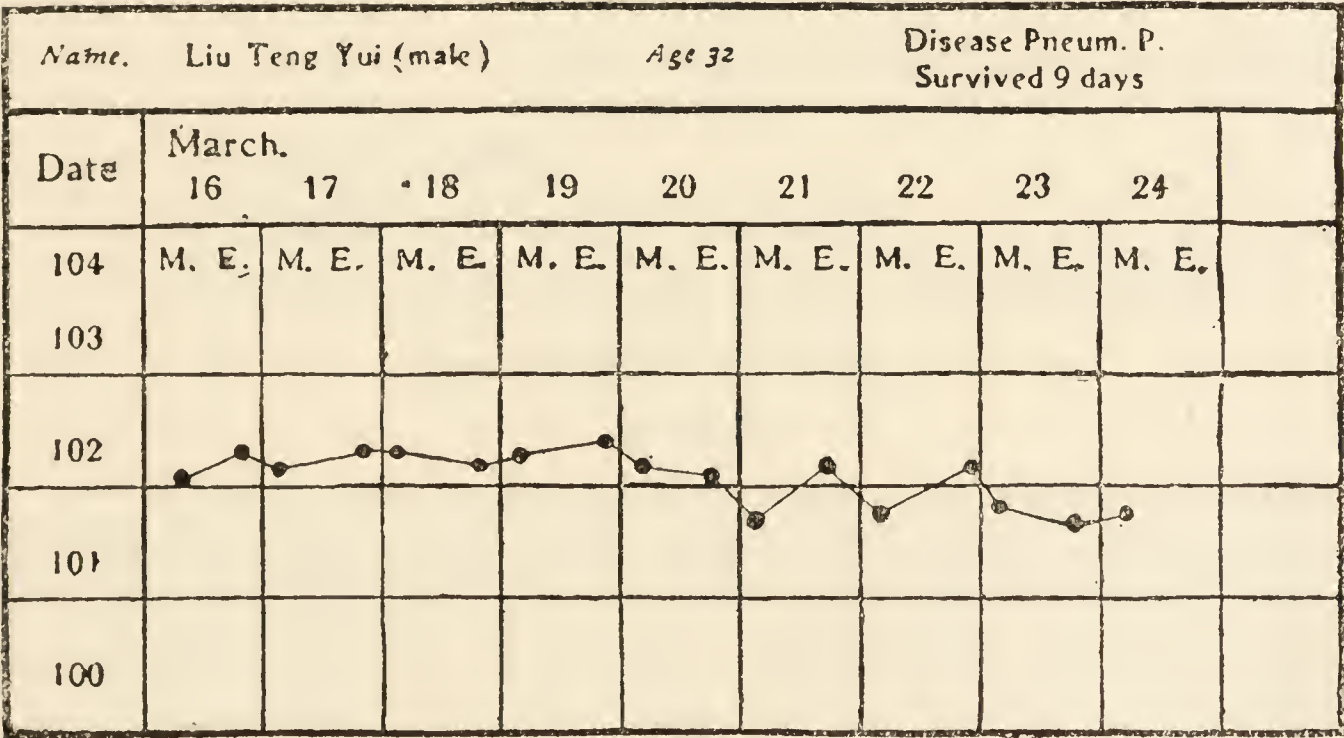
TABLE IV

Tabulation of our Sanitary personnel in Dalainor

Character.	No. employed.	No. infected.	% infected.
Doctors	3	0	0.0
Dressers	5	0	0.0
Sanitary coolies ..	10	5	50.0
Sanitary police ...	40	3	7.5

Note:—The original sanitary personnel in Dalainor were vaccinated with plague vaccine, including 6 of the 10 coolies (four of those vaccinated died) and all the policemen.

TABLE V



PATHOLOGICAL FINDINGS IN PLAGUE PNEUMONIA, SECOND MANCHURIAN EPIDEMIC 1920-21

*(From the Laboratory of the Manchurian Plague Prevention
Service, Harbin)*

BY

WU LIEN-TEH, J. W. H. CHUN, AND ROBERT POLLITZER

CONTENTS

I. INTRODUCTION

- a. General
- b. Technique

II. CLASSIFICATION OF CASES (ACC. TO LUNG FINDINGS)

- Group A. Cases with marked Pneumonia and Pleuritis (19)
- Group B. Cases with slight lung and pleural changes (5)
- Group C. Cases without Pneumonia or pleural changes (9)

III. OBSERVATIONS

IV. FINDINGS IN OTHER ORGANS

V. SPECIAL CASE OF MOTHER AND STILLBORN

I. INTRODUCTION

i. General

In reporting on the macroscopic findings of the *post mortem* examinations performed during the second Manchurian Plague Epidemic of 1920-21, we do not deem it necessary to begin with an extensive review of former publications dealing with Pneumonic Plague, as such has already been done by Strong, Crowell and Teague ⁽¹⁾, as well as by Wu Lien-teh and Woodhead ⁽²⁾ in an exhaustive manner. It seems advisable, however, to cite some of the findings observed during previous epidemics in order if possible to clear up questions which are at present unsettled and to emphasize any similarities or

(1) Philippine Jl. of Science, Vol. VII. Sec. B. No. 3, 1912.

(2) British Jl. of Pathology and Bact. 1912.

discrepancies seen by us on this occasion as compared with observations made previously by others. These points will be mentioned under their several headings. In this article, only the gross pathological findings will be described, while the histological features will be reserved for a future contribution.

Although both Childe (1897-98) ⁽³⁾ and Albrecht and Ghon (1897) ⁽⁴⁾ have carefully described the *postmortem* appearances as found by them in twelve and three cases respectively of Pneumonic Plague, these must be regarded as isolated instances in the chain of Bubonic epidemics generally encountered in India. It was not until the great Manchurian outbreak of 1910-11 occurred that these purely pneumonic cases could be studied wholesale. During this period, Strong and Teague ⁽⁵⁾ performed 25 autopsies at Mukden, Fujinami ⁽⁶⁾ made 29 sections (including three on animals) at Mukden, while Koulecha ⁽⁷⁾ described the results of 28 necropsies as made at Harbin. A quiet interval of ten years then supervened in Manchuria, after which the second epidemic occurred beginning in October 1920 at Hailar and ending in May 1921 at Harbin, although further east, such as Vladivostok, (488 miles east of Harbin) the final case was reported as late as the end of October 1921. Together with two Russians colleagues our Chinese Medical Officers made sixteen autopsies at Hailar (467 miles west of Harbin) during the earlier part of this epidemic, while Dr. Hsieh of our Service made three *postmortems* on plague cases at Dalainor (566 miles west of Harbin) but no complete records were kept of these cases, and most of the specimens were damaged or spoilt during transit. At Harbin, thanks to the superior facilities offered by our main laboratory, we were able to perform forty-three complete autopsies, of which thirty-four proved to be true pneumonic cases. The other nine non-plague bodies belonged either to cases admitted into our infectious wards during the epidemic or to cadavers picked up in the street under circumstances suspicious of plague.

The principal viscera of the plague series have been preserved in Kaiserling solution for further observation whenever needed. The whole respiratory organs from the tongue downwards together with oesophagus and heart were removed *en masse*. Careful notes were made by two of us immediately after each *postmortem*, and these were checked on a second and on a third occasion with the Kaiserling specimens, additions or corrections being made wherever necessary.

(3) British Med. Jl. London, 1897, I, 1215; 1898 II, 858.

(4) Ueber die Beulenpest in Bombay im Jahre 1897, Wien, 1900.

(5) Philippine Jl. of Science, Manila, Vol. VII. Sec. B. N. 3, 1912.

(6) Report of International Plague Conference, Mukden, 1911, P. 144.

(7) Report of International Plague Conference, Mukden, 1911, P. 151.

With the exception of the ones picked up from the streets in May, practically all the bodies upon which *postmortems* were made were fresh, having been chosen from cases which had died a few hours previously. Even in the case of those where twelve or more hours had intervened, no signs of decomposition were noticed, as the months of February and March were cold, the temperature being seldom above 0° C. The hospital crematorium was situated about fifty yards from the *postmortem* room, both lying in the same compound, so that no undue effort was required to transport a body after section from the *postmortem* room to the cremating pit. All dead bodies from the plague wards were cremated in the hospital crematorium.

ii. Technique Employed

It may be interesting to mention some points in connection with our *postmortem* technique when dealing with plague cases in the midst of winter. The body to be examined was brought on a wooden stretcher by two attendants. Only four persons were present in the room—three doctors and an assistant. In addition to our usual two-tailed cotton and gauze respiratory mask, goggles were worn, but these often proved inconvenient owing to the condensation of water upon the glasses when coming in from the cold air, and were sometimes discarded. Long india-rubber boots were worn besides the usual overalls. Every one was provided with a pair of medium weight rubber gloves except the actual operator who had an extra pair of long thick ones over the inside pair. In this case, the cuffs of the gown were tied round the wrists after the first pair had been put on; the long pair was next adjusted and tied on the fore-arm. The fingers and wrists thus had a double protection. To further minimise the possibility of accidents, only one doctor performed the actual section and removed the organs, while the others assisted with the trays, pails, liquids, smears and cultivations. After the section was finished, the body was wrapped up in a large piece of cloth which had been soaked in strong cresol solution, and tied at the neck, around the chest and extended arms, and hips. It was then placed on the stretcher and taken to the cremation pit by the attendants. All the instruments, gloves and trays were placed in a large pail and boiled by the P. M. room assistant.

II. CLASSIFICATION OF CASES

Of the 34 plague cases examined at Harbin, there were 19 with marked pneumonia and pleuritis; five cases showed only slight pneumonia and no pleuritis, thus forming a transition to a third group of nine cases in which no pneumonia could be found. The one remaining was a unique autopsy, being that

of a full-time stillborn foetus of a plague infected mother. This last will be described in a separate chapter. Summarised, these 34 cases run as follows:—

A.	Extensive pneumonia and pleuritis present	19
B.	Slight pneumonia and no pleuritis present	5
C.	No pneumonia and no pleuritis present	9
D.	Special case of stillborn	1

GROUP A. CASES WITH MARKED PNEUMONIA AND PLEURITIS (19)

Of these nineteen, ten were of male adults, six of female adults, and three of male children.

Tongue, Fauces and Pharynx.

Our results in the main are in accord with those noted in the 1910-11 epidemic, the tongue being swollen, brown at the anterior portion, often dark-red and sometimes haemorrhagic at the base, while the pharyngeal wall was congested. In a few cases, only slight hyperaemia and swelling of the papillae at the base of the tongue were noticed. The tonsils were swollen in thirteen of our 34 cases, including P.M. 36 which was complicated by an old abscess. Some congestion was generally seen on the surface and on section. In a few we were able to squeeze out drops of purulent matter on cutting the substance, but until the histological examination proved positive, this could not be regarded as pathognomic of plague. This point will be referred to again in Group C. of our cases.

Larynx, Trachea and Bronchi.

As a rule, the whole respiratory tract from the fauces and larynx downwards assumed a progressively red colour, in some cases not so marked as in others. The blood vessels were injected. On many occasions the epiglottis was intensely swollen and purple in colours. The larynx and vocal cords were generally red and swollen. The trachea was usually covered with pink blood-stained mucus or froth, and its walls were injected, small haemorrhagic points being now and then found in the mucous membrane. This inflammatory change was generally most marked at the bifurcation. We have five cases on record, however, where the larynx and trachea were but slightly congested, so that the mucosa showed only a pink colour. In a sixth case, the trachea presented but a slight reaction, and bloody froth was seen only at the bifurcation. In two instances, simple whitish froth was encountered mixed in places with greenish matter. In one case there were haemorrhages not only in the trachea but also beneath the mucosa of the larynx. In two more, haemorrhages were present

in both epiglottis and larynx. One of the latter showed some small patches of necrosis in the mucosa of the trachea. The bronchi, large and small, were markedly congested along the whole mucosa. So far as we could see in no case did this congestion appear to become less marked as the smaller tubes were reached. The deep red longitudinal stripes in the bronchi near the bifurcation as described by Strong ⁽⁸⁾ could be seen quite frequently.

Pleura.

In our series, we rarely noted ecchymoses beneath the parietal pleura, which were seen often, especially by Strong, in the first Manchurian outbreak, but if seen they were most often situated on the diaphragm. In one instance, we saw an unusually intense congestion of the parietal pleura.

Fluid in the pleura was met with in four of these pneumonic cases, in one case it was blood-stained, in the others greyish-white. It must be stated that while doing spleen punctures for diagnostic purposes, we found not infrequently the spleen dislocated by an exudate situated apparently in the left pleura.

In comparatively few necropsies did we find any fibrin threads or membranes on the surface of the lungs, although these appeared rather common in former epidemics.

In one case we found an inflamed area covered with a fibrinopurulent layer, in three cases with a purulent layer. Microscopic preparations of this purulent matter showed numerous plague bacilli in the leucocytes as well as outside.

Lungs.

All cases in this group had at least one pneumonic area showing the characteristic acute inflammatory changes in the pleura and adjacent lung substance which form such a prominent feature in pneumonic cases. The changes met with most frequently were thickening of the visceral pleura over the whole or part of the surface of a pneumonic area, the pleura being yellowish, with more or less confluent haemorrhages underneath. As stated before, we did not find this characteristic change on every pneumonic area involving the surface of the lung. In few instances only moderate changes were seen. Among these could be noted some haemorrhages over the slightly bulging pneumonic areas, with no pleural changes and no haemorrhages elsewhere on the pleura. Again, while at times haemorrhages could be detected beneath the whole pleura, on the other hand in two cases we noted haemorrhages over circumscribed areas which were not involved by pneumonia. In others the pleura over a pneumonic area was

(8) Philippine JI. of Science, Vol. VII, Sec. B, No. 3, P. 215.

cloudy, sometimes slightly injected, sometimes showing more or less confluent haemorrhages underneath, but it was seldom thickened. In some pneumonic patches we observed beneath the markedly changed pleura the "yellow and red dots and spots" as described by Albrecht and Ghon.

At this stage it may be advisable to refer to former descriptions of the lung changes observed in plague pneumonia, as the different authors did not seem to agree, thus making it difficult to obtain a standard for purposes of comparison.

Whereas some of the earlier workers like Wyssokowitz and Zabolotny⁽⁹⁾ Albrecht and Ghon, and Childe agreed that the pneumonic areas or patches found in their cases were of the lobular or catarrhal type with a tendency to become confluent, Strong and Fujinami stated that the pneumonic areas met with in their *postmortems* were frequently of the lobar type. The report of the Anglo-Indian Plague Commission (1900, 1901) also arrived at the same conclusion. For instance, Strong stated that "although it has been proved that the pneumonia is primarily bronchial in origin or of the lobular type, nevertheless very early lobar involvement was very much more frequently encountered....". In describing his findings in detail, Strong emphasized that he never observed grey hepatisation of a whole lobe, also only rarely large areas of red hepatisation, but that he had cases in which he saw in the same lobe both red and grey hepatisation, whereas another part of the same lobe showed only engorgement. He found both types not only in the same cases but also apparently in the same lobes. On the other hand, in his concluding remarks, he said: "the bacilli rapidly multiply and produce at first pneumonic changes of the lobular type and shortly afterwards from the fusion of several rapidly spreading areas more general lobar involvement of the lung tissue."

From this we may gather that Strong distinguished between the "lobular" and "lobar" form of plague pneumonia only so far as the extension of the pathological processes is concerned, but that he considered the latter form as a result of a bronchopneumonia which had become confluent.

Be that as it may, we ourselves find it impossible for the time being to classify all of our cases into the lobular or lobar type of pneumonia. Whereas a considerable number of the changes observed must be classified under the first group, a small number have a lobar appearance while there are quite a number of intermediate forms in which it would be difficult to decide one way or the other. We hope that after a thorough

(9) Ann. de l'Inst. Pasteur, 1897, XI, p. 663.

histological examination it will be possible to make more definite statements and to clear up not only the nature of our cases but to a certain degree this whole problem of plague pneumonia.

Before entering into a detailed description, we might state that, as noted by former observers, all the cases of this group presented marked congestion and oedema of the lungs. Should any superficial pneumonic focus exist, a corresponding bulging of that area of the lung surface would be seen. As for the seat of hepatisation, it can be seen from Table I that out of 19 cases, 6 showed involvement of one lobe only (one right lobe in two cases and one left lobe in the other four), 3 showed involvement of two lobes (right lobes twice and right and left lobes once), 2 showed involvement of three lobes (all the three right lobes once), 5 showed involvement of four lobes, and the remaining 3 showed involvement of five lobes. So that in five of our cases the right side alone presented pneumonia and in four the left side alone.

As to the distribution in the different lobes, the right upper lobe was involved 13 times, the right middle lobe 7 times, the right lower lobe 12 times, the left upper lobe 10 times, and the left lower lobe 11 times.

According to our observations the right middle lobe was more often involved than in Fujinami's list, but in general the distribution in our cases agreed with his findings. When there was only one pneumonic area present, it was never found in the right middle or in the right lower lobe. It was situated twice in the right upper lobe, once in the left upper lobe and 3 times in the left lower lobe.

The frequency and distribution of the different forms of pneumonia about to be described can be seen in Table I.

The most frequent pneumonic change noted was that of a confluent lobular pneumonia. Often there were lobular foci in the stage of red hepatisation which became confluent to form small but in some instances large pneumonic areas. But in the latter case, the whole appearance of the area with islands of non-hepatised lung tissue was sufficient to confirm the diagnosis.

In a few instances, especially when a large portion of a lobe was affected, the colour of the pneumonic areas was reddish-grey or purely grey in part. In one case, almost the whole affected tissue had a reddish-grey colour. This case

(P. M. 23) is also remarkable because it can be clearly seen how the different confluent areas are arranged round the ramifications of a small bronchus, thus forming a picture similar to, though of a more advanced type than, that described by Strong. It is hardly necessary to state that in using the term "lobular confluent pneumonia" we do not mean that the changes thus denominated are the same as those met with as secondary lung affections associated with other acute infectious diseases. It would be better, of course, if there existed a special nomenclature for the changes found in plague pneumonia, but as it is, we must use the general terms of morbid anatomy of the lungs for the characteristic changes which are so pathognomic of plague pneumonia.

In about half of our cases, other manifestations of pneumonia were noticed, namely more or less well-defined patches which showed a homogenous surface on section without islands of hyperaemic areas, and were situated most often at the periphery but sometimes in the central part of the lung. As a rule, changes in the lung and bronchi were in no way different from the other cases. The pleural changes were the same as those met with in connection with the lobular confluent areas. We are using purposely the term "more or less well-defined" for these areas, because in our cases we could only now and then note the distinct ring of engorgement described by Childe and Strong. We rarely found the wedge-shaped patches described by Strong. In every other respect however they were apparently similar to those patches described heretofore. The colour in our cases, was often red or greyish red, but sometimes the central portion or whole of the superficial patches was grey.

In 11 of the cases of this group we found more extensive changes in the lungs than those described above. Among these eleven cases three exhibited such changes as the only manifestations of plague in the lungs. In other instances, they were found in combination with the forms of plague pneumonia described above. Rarely, both forms were present in the same lobe. In only two instances were extensive changes noted in two lobes of the same lung. With two exceptions where they were localised in the left upper and left lower lobes, these were found in the right lung. The upper lobe alone was affected five times, the middle lobe alone twice; both upper and lower lobes once, and both middle and lower lobes once. The accompanying table may simplify matters in this respect:

LOCALISATION OF EXTENSIVE PNEUMONIC AREAS IN GROUP A (11 CASES)

No. of P.M.	Sex	Rt. U.L.	Rt. M.L.	Rt. L.L.	Left U.L.	Left L.L.	Remarks
1	M	1	
2	F	1	
5	F	...	1	1	Only very small part of the lobe free.
6	F	...	1	Only very small part of the lobe free.
10	M	1	
12	M	1	Only very small part of the lobe free.
13	F	1	
19	F	1	...	1	Only very small part of the lobe free.
21	M	1	...	
23	M	...	1	
27	M	1	

In none of our cases was one lobe completely affected. In four only small portions of the lobes were free from hepatisation. The site of hepatisation in these was distributed as follows:—

(a) right middle and right lower lobes (P. M. 5); (b) right middle lobe (P. M. 6); (c) left lower lobe (P. M. 12); and (d) right lower lobe (P. M. 19). In one of the above cases (P. M. 5), the whole affected area of the middle lobe indicated a stage of grey hepatisation, and stood out fairly well-defined against the adjacent small congested portion. In the three remaining ones, only partially grey hepatisation was present and it was impossible to draw a sharp borderline between the pneumonic and congested parts. In all the other cases, although the pneumonic areas were sometimes rather extensive, a considerable portion of the lobes was free from pneumonic

changes. This stood out either in substantial congested masses against adjacent solid areas of pneumonia, or in the form of small tracts of congested tissue unevenly distributed at the margin among the larger hepatised areas.

A whole extensive area in the stage of red hepatisation was seldom present in our list of necropsies. Sometimes grey hepatisation predominated. Sometimes when both stages were present, a zone of grey hepatisation might be seen surrounded by one of red hepatisation. None of the eleven cases differed in other respects from the lobular pneumonic type, with the exception of P. M. 23 which showed an extensive pneumonic area in the right middle lobe. This case also presented a picture which might be mistaken for the lobar type inasmuch as it showed small ill-defined confluent greyish-red areas and a bigger well-defined grey area bordered by a smaller focus of red hepatisation. It did not exhibit the usual pleural changes, however, being covered with a firmly adherent layer of fibrin, beneath which was a whitish thickened pleura devoid of haemorrhages. This condition of the pleura seems quite unique.

Among the female plague cases, such extensive changes as described above were more prominent. This matter will be referred to later on.

Bronchial glands.

In every one of the pneumonic cases we found more or less marked acute changes in the bronchial glands. Although these corresponded in the main with the descriptions given by Strong and Fujinami, it must be stated that in our cases the macroscopical changes did not seem as marked as these noted by the former. A large majority of the glands showed extensive anthracosis, while petechial haemorrhages were in some instances observed. The tracheal glands were sometimes swollen and congested.

The question of chronic tubercular changes in the lungs and bronchial glands will be treated under a separate heading.

The changes in other organs will be grouped together, as only differences of a minor nature were found in the three groups.

GROUP B. CASES WITH SLIGHT LUNG AND PLEURAL CHANGES (5)

Although we do not profess to state that the five cases described here are widely different from those recounted in Group A, yet they seem to form a connecting link with the cases of the next group and may therefore be treated separately. Of these five cases, the pneumonic changes in two instances

were those of a lobular confluent character, while in the other three they were in the form of patches, which were situated peripherally twice and centrally once. The pneumonic areas in these cases were smaller and less well-defined than those mentioned in group A. The visceral pleura remained apparently unchanged. In two instances, some haemorrhages were seen beneath the pleura and away from any pneumonic area. In one case (P. M. 4) a considerable quantity of non-sanguinous fluid containing numerous plague bacilli was found in both pleural cavities. In P. M. 17 the pleura did not show any change, but over both lower lobes a circumscribed area of a fine fibrin net was present while haemorrhages were seen over the pneumonic areas. In this specimen also, white irregular areas of broncho-pneumonia could be distinguished in both upper and lower lobes easily visible from the surface of the lungs. Hence this fifth case may be said to possess unusual features.

Regarding the other organs, in two cases the respiratory tract presented the usual swollen and congested appearance. In the other three (P. M. 4, 17 and 31), the changes were noticeably less prominent. The mucosa of the larynx and trachea had only a pink colour, there was no bloody froth or sputum but only mucopurulent matter in two cases (P. M. 17 and 31). In P. M. 4 the bronchial mucosa was not as congested as usual. The bronchial glands in all the cases except one adult man (P. M. 31) presented a more or less congested appearance. In this adult case there was considerable swelling due to old caseating masses with no sign of acute changes.

GROUP C. CASES WITHOUT PNEUMONIA OR PLEURAL CHANGES (9)

These nine cases (boy of two years and 8 adults) showed congestion and frequently oedema in the lungs, but no signs of any consolidation due to pneumonia could be found.

Smears taken from the lung substance showed only a few plague bacilli. In one of these cases (P. M. 35) both smears and cultures from the lungs proved negative for *Bacillus pestis*, while the spleen gave abundant growths of the organism. The pleura in all nine cases was apparently normal; in only two cases could some small haemorrhages be seen beneath the visceral layer.

The upper respiratory tract presented the usual swelling and congestion in only three of the nine cases recorded, while in the remaining six these changes showed slighter variations. No bloody sputum or froth was seen in the larynx or trachea in any of these nine necropsies. Either no contents at all

were encountered or, as in four instances, the mucus or froth present was not bloodstained. Smears taken from the trachea, with one exception (P. M. 39, where many plague bacilli were found in the scrapings from the almost dry trachea), showed very few plague bacilli, thus forming a striking contrast to the findings in those cases displaying marked changes in the respiratory tract. Similarly, the macroscopic changes in the bronchial glands were mostly of a trivial nature. The mucous membrane of the bronchi was in every adult case swollen and congested. In the child (P. M. 14) the changes in the bronchi were less marked than usual, though more than in the upper part of the tract. On the other hand, no changes were present in any of these cases, which might suggest another mode of entry of the organism, such as through the tonsils, or the digestive tract. The general absence of buboes in the cervical, axillary, inguinal and femoral regions was particularly significant. Nor could these be found in a large number of unautopsied corpses brought to the grounds of the hospital during the later weeks of the epidemic although splenic punctures supplied ample evidence of plague. One apparent exception should be recorded, namely, a case where the cervical glands on both sides were swollen; in this, one gland dissected out measured the size of a hazelnut. In this same case (P. M. 36) we noticed a small abscess in the substance of the right tonsil, containing thick pus, but no other changes were observed similar in appearance to the extensive invasion of the fauces as described in one of Fujinami's cases, nor was there any oedema of the glottis as pointed out by Albrecht and Ghon. Smears taken from the tonsillar pus showed but a few plague bacilli mixed with other microorganisms. Neither did our histological examination of this case supply any evidence of primary tonsillar infection.

So Fujinami's case seems to be the only authentic case of tonsillar infection observed in the Manchurian outbreaks, as Koulecha's findings were doubted by all the other authorities.

We regret our inability to report clinically on the cases of this group C. as with one exception the autopsies were performed on unidentified cadavers brought to us from the streets. At that time no fresh plague cases were admitted into the hospital. The exception just mentioned was a man 25 years old (P. M. 39), admitted into hospital the day before his death as a suspected plague case. He had no cough or expectoration, only high fever and weak pulse. The *postmortem* done on the day of his death showed the same general changes as met with in cases of this group, only there were more plague bacilli in the smears from the trachea, as already stated above.

But as this patient did not cough or spit, the presence of a larger or smaller number of plague bacilli in the trachea may not be of special importance from an infective point of view.

Although the histological examination of these cases is not yet complete, there is no doubt that the mode of infection in them was the same as in those cases exhibiting pneumonic lesions, and that between the two groups there exists only a gradual and not an abrupt demarcation.

The above points seem to establish the fact that, apart from the true pneumonic cases accompanied by more or less marked changes in the respiratory passages there exist not a few cases, where, in addition to the absence of inflammatory changes of a pneumonic character in the lungs, the fauces, larynx, and trachea also show only a slight reaction.

In other words, although the method of entry of the *B. pestis* in this Group C. as in the Group A. and B. was through the respiratory channel, leading in all cases finally to plague septicemia, at least nine out of total 34 cases (i.e. 26.5%) did not show pneumonic lesions of any sort. Hence the term Pneumonic Plague or Plague Pneumonia as generally applied to this disorder is not strictly accurate, and should be replaced by "Pulmonary Plague" as suggested by one of us in 1911 when acting as President of the International Plague Conference, Mukden ⁽¹⁰⁾. The expression "Pulmonary Plague" would then rightly embrace all the features—non-pneumonic as well as pneumonic—present in this affection.

III. OBSERVATIONS

These pulmonary cases in the strict sense appeared with scarcely any exception towards the end of the epidemic (see Table II) and in our opinion contributed not a little towards its termination. It may be asked why we came to this conclusion. In the first place, because of the absence of sputum and apparently of cough, these cases during life were far less infectious than those in Group A and B. What factors were responsible then for this evolution of the pneumonic type into the pulmonary type? The first thing to strike one's mind would be a diminished virulence of the organism due to the approach of warmer weather, but this is discounted by three objections: (1) Scientific evidence brought forward at the Mukden International Plague Conference 1911, (2) the prevalence of the epidemic throughout the summer in Vladivostock (May-October 1921) and (3) our own experience of the undiminished virulence of the strains cultivated during the 1921 epidemic and used for animal experiments throughout the summer months.

(10) Report of International Plague Conference, Mukden, 1911.

It is possible that this transformation from the pneumonic to the pulmonary type might be due principally to the *enhanced* virulence of the *Bacillus pestis* at the height of the epidemic, so that the patient succumbed before any gross anatomical changes could develop. In fact the organisms having successively passed through their human hosts were becoming so virulent, that there seemed to be little or no time for the latter to develop pneumonia. As a consequence the principal medium of infection, namely the sputum, was absent.

Again, the later victims became less infective though invaded by more virulent bacilli, and fewer infections therefore took place, which were apparently of the pulmonary type described above. These pulmonary cases proving less and less infectious, the epidemic gradually withered away.

The one exception showing a pulmonary and not a pneumonic lesion which occurred in the middle part of the epidemic was that of a two year old child (P. M. 14). In this case, the tongue, larynx, and trachea showed very slight reaction, while the lungs were oedematous and congested with no pneumonia. This condition could be explained by there having been insufficient time for the development of pneumonia owing to the high toxicity of the invading organism and the diminished resistance of the patient. Should any future epidemic of plague pneumonia occur, this question of differentiation between the pneumonic and pulmonary types might be more fully investigated. In this connection we may state that we propose to continue our inhalation experiments upon tarabagans and other animals in 1922 with a view to the further elucidation of this rather obscure problem.

IV. FINDINGS IN OTHER ORGANS

The morbid anatomy of the remaining organs in all these groups comprising 33 cases (the stillborn excluded) may be conveniently described together, as there are no marked differences between them in this respect. Our findings in this epidemic confirm on the whole those of the 1910-11 outbreak, hence this part of our report will be necessarily brief.

Pericardium, Heart and Bloodvessels.

We noted in 11 of our cases (almost exclusively those with marked pneumonic changes) the presence of fluid in the pericardial sac. In one of these, there were almost 400 c.c. of clear fluid in the sac, dislodging the heart considerably from its normal position. As in the case of pleural exudate, the pericardial fluid was rarely tinged with blood. It was sometimes cloudy, though more often it remained clear, and plague bacilli could in most cases be cultivated as well as seen in

smear preparations. We detected haemorrhages of varying size in the parietal layer in ten, and in the visceral layer in nine out of 34 cases. In only four instances were haemorrhages found in both parietal and visceral layers of the same pericardium.

The right chamber of the heart was in almost all cases distended with blood, the right auricle being often quite thin from dilatation. In one case (P. M. 11) the heart was unusually contracted. The cardiac muscle was invariably softer than usual, and indicated cloudy swelling and sometimes early fatty degeneration. The large bloodvessels showed little or no acute change. In P. M. 4 (child of 3 years) some petechiae were noticed beneath the intima of the tricuspid valve.

Alimentary Tract.

Contrary to previous observations, we not infrequently found some slight congestion in the oesophagus. Out of 33 cases recorded 23 were normal. Of the remaining ten, seven showed slight congestion in the lower third, one in the middle, one in the upper part, while the tenth was hyperaemic along its whole course. The pharynx being practically always affected, the sharp line of demarcation between this congested part and the pale oesophagus was quite marked.

The mucosa of the stomach was often slightly swollen. Haemorrhages of varying extent were frequently present, but no other acute changes could be detected.

With regard to the intestines, the peritoneal coats showed haemorrhages in only a few instances. Hyperaemia was often noted in the mucosa of the small and large intestines. In some cases both coecum and ileum showed ecchymoses in the submucosa. Cultures of *B. pestis* were successfully obtained from the congested areas of the intestinal mucosa in two instances, while the faeces from the coecum in another case gave positive smears. The mesenteric glands in over 21% of the cases were swollen and congested. The parietal layer of the peritoneum was generally free, but in one case (P. M. 21, aged 61) we noted both congestion and haemorrhages in this region.

Spleen.

The spleen was found distinctly enlarged in 71% of our cases as compared with 56% of Strong's. In a further 19%, slight enlargement was recorded. Although the spleen was usually firmer than in other infectious diseases, as pointed out by Strong, in a quarter of our cases the organ was soft and flabby, even breaking into two or more pieces when extracted from the abdominal cavity. This flabby condition and increased size of the organ were apparently more marked in the later

pulmonary than in the earlier pneumonic cases. Until the histological examination is completed, it would perhaps not be advisable to over-emphasize these features. Haemorrhages beneath the capsule were seen in two cases. When cut, the splenic pulp was found to be darker than normal, while the follicles were visible in at least half of the cases. In one case (P. M. 11, man who died after an illness of nine days) a small white infarct measuring 7-8 mm. across was found at the lower border of the spleen.

Liver.

Cloudy swelling or distinct fatty degeneration was noted in all cases. In about half of these, we observed lying beneath the capsule yellowish white patches, more or less circumscribed from the rest of the tissue. These patches varied in size from $1\frac{1}{2}$ to $2\frac{1}{2}$ cm. on the surface, while on section they measured 1 to 2 cm. deep. They were very rarely situated in the interior of the organ away from the surface and were apparently the results of acute toxic changes produced by the *B. pestis*. The patches varied in number, several being often found in the same organ.

In three cases the liver presented a nutmeg appearance. The microscopic examination will ascertain whether this is due to "acute degeneration of the liver cells around the hepatic veins, and not to a chronic venous congestion" (Wu and Woodhead). Haemorrhages beneath the capsule of liver were seen in 8 out of 34 cases, 5 being pulmonary and 3 pneumonic.

Pancreas.

This was rarely swollen, only slight hyperaemia being seen in a few cases. In one case some haemorrhages were noted in the substance of the organ.

Lymphatic glands in general.

Beyond the changes already noted in the bronchial, tracheal and mesenteric glands, nothing of an unusual nature was found in the other lymphatic glands. In a few cases, there was slight enlargement and congestion of the glands of the neck. The unusual case of a distinct cervical swelling found in P. M. 36 has already been mentioned.

Thyroid, Thymus and Adrenals.

No peculiar changes were seen in the thyroid, except slight congestion in a few instances.

In the case of the thymus, we were interested to note that in more than half of our cases the organ was persistent in the adult bodies, sometimes reaching a remarkable size. But inasmuch as this gland was apparently persistent also in a

smaller, though a fair percentage, of non-plague bodies examined by us, it will perhaps be advisable not to lay undue stress upon this phenomenon, the more so as recent literature conveys warnings not to exaggerate the importance of the *status thymicus*. The matter, however, deserves further study by both anatomists and pathologists in China.

The adrenals presented no marked naked-eye changes. The centre of the gland was sometimes found empty so as to produce a *cul de sac*, but no special importance need be attached to this point as it is present in a majority of normal autopsies.

Urinary and Sexual Organs.

Our findings in the kidneys corresponded closely with those of former observers. The cortical bloodvessels were often considerably dilated. Actual haemorrhages were seen on seven occasions beneath the capsule and on eleven occasions in the pelvis of the organ. The urinary bladder also showed haemorrhagic points in a few cases. Cultures taken from the cavity of the pelvis, ureter and bladder (the latter drained in some instances by catheter) gave as a rule positive results for *B. pestis*. On the other hand, with one exception, urine voided by thirty living patients proved negative for this organism, when cultivations were made. No further changes beyond congestion were apparent in the male and female sexual organs. Positive cultures were obtained in some instances from the testis as well as the vaginal portion and the cavity of the uterus.

Central Nervous System.

The brain showed marked congestion, the subdural vessels being often enormously dilated. Oedema of the cerebral hemispheres was sometimes present. The cerebrospinal fluid was clear and gave as a rule positive bacteriological findings. The petrous portion of the temporal bone was removed in several cases for more minute investigation.

Tuberculous Lesions found in Plague Cases.

The incidence of plague in tuberculous subjects was specially discussed at the Mukden Plague Conference (1911). Strong made 25 complete necropsies at Mukden and did not find a single tuberculous lesion. Fujinami performed 26 human plague *post-mortems* and found a few lymphatic glands affected with TB and only one case where the lungs showed any TB. lesions. Koulecha made 28 autopsies and did not mention TB. Haffkine said he found calcareous nodules in 3 cases of plague pneumonia.

In view of the apparent rarity of Tuberculosis among plague-victims, special care was taken by us to ascertain its presence in our cases. The following are the findings among 34 necropsies:—

a. Lungs.

	Marked Pneum.	Slight Pneum.	Pulmon.	Cases.	Total.
	Group A.	Group B.	Group C.		
Active lesions	1	—	—		1
Latent „	4	—	1		5

In the only active case seen—a well-developed but not fat old man of 61—there was no cavitation, only a confluent granular tuberculosis of the right apex with many calcified nodules in other parts of the same lobe. The case was evidently one of benign character, often seen in persons of advanced age.

The five latent cases showed only induration and calcification and no recent changes.

b. Peribronchial Glands.

	Marked Pneum.	Slight Pneum.	Pulmon.	Cases.	Total.
	Group A.	Group B.	Group C.		
Acute changes	1	1	—		2
Latent „	2	1	—		3
Doubtful	—	—	1		1

The two acute cases showed distinct caseation throughout the affected glands, while the latent cases had some calcifications. The doubtful case appeared to possess a few suspicious areas of softer consistence. These will however require the help of the microscope before a definite conclusion can be reached. It is quite possible that such an enquiry will reveal further cases at present undetermined.

c. Pleural Adhesions.

	Marked Pneum.	Slight Pneum.	Pulmon.	Cases.	Total.
	Group A.	Group B.	Group C.		
Extensive	4	1	5		10
Slight	6	1	1		8

d. Changes in Other Organs.

- i. One case of scrofuloderma over lower part of chest was seen (P.M. 7).
- ii. One case had rather extensive adhesions in the upper part of the peritoneum (P.M. 29).
- iii. Over the front of the epicardium in another case were seen two milk spots (Sehnenflecke) each the size of a sixpenny piece. It is possible this may be of rheumatic origin.

Remarks.

To summarise, we had four cases of active Tuberculosis (one in lung, two in glands and one in skin) in addition to a doubtful gland case. 18 cases of pleural adhesions were noted, apparently tuberculous. The above findings were distributed among 20 cases altogether.

Compared with our usual out-patient records, where the percentage of active tuberculous cases was considerable (for

instance, Harbin Hospital reports for 1918-21 enumerated 993 of TB lungs, 926 TB skin, 580 TB bone and 3050 TB glands, i.e. a total of 5549 TB cases out of 39,819 patients treated) this scarcity of tuberculous lesions in plague victims may seem somewhat inexplicable, unless we take into consideration the following factors:—

- I. Plague patients came principally from the strong ricksha pullers, day coolies, etc., who were crowded in dusty dark inns, where they received plague infection.
- II. Patients suffering from chronic complaints, like Dysentery, Phtisis, etc., were often turned out of the inns during the epidemic and sought admission into our general wards. When these died no *postmortems* were made from them.
- III. Our plague autopsies were performed upon patients who had died in the Hospital up to the end of the epidemic when, owing to the absence of admissions into our hospital, all dead found in the streets were carted into the hospital compound, and the necessary necropsies made for diagnosis. Among the latter cases many morphine subjects were found, who at the *post mortem* revealed many pleural adhesions.

It seems therefore inadvisable to lay undue stress upon the comparative absence of acute tuberculous lesions among plague cadavers.

Incidence of Age and Sex (see Table II).

The only *postmortem* performed upon an old person was P.M. 21, aged 61, showing marked pneumonia and pleural changes. In this aged subject, we noticed no striking changes in the viscera as compared with those in younger bodies.

We autopsied, beside the stillborn, the bodies of 6 children below ten years. Attention has already been called to three of these. The other three children also exhibited no unusual features.

Regarding the six adult females, all showed considerable pneumonic and pleural changes and were hence classed under Group A. In five, extensive pneumonia was present. No other differences were noted.

V. SPECIAL CASE OF MOTHER AND STILLBORN

a. A woman aged 37 years, pregnant for the fourth time, was sent to hospital with fever as a suspect on morning of 22nd March from the isolation wagons, where she was lodged after her husband had been attacked by plague. When admitted into hospital, she was coughing up blood, in which plague bacilli were found. In the afternoon, the patient became worse and towards evening gave birth to a full-term female child. The mother died early next day. The *postmortem* upon the mother showed plague-pneumonia, with the following summary of findings:—P.M. 9, woman, mother of stillborn, died 23rd March, 1921. P.M. soon after.

Well built woman, age 31. Cyanosed face.

Tongue slight red; *Tonsils* swollen and congested. *Papillae* of *Pharynx* show similar changes.

Larynx and *Trachea* much congested, little mucus.

Lungs. Anterior surface showed film of semipurulent mucus, emphysematous.

Right upper lobe pneumonic patch. Bronchopneumonia in right lower lobe. Rest congested.

Left lung markedly congested, no patches.

Peri-bronchial glands swollen and anthracotic.

Heart. Much clear pericardial fluid. Much enlarged. Coronary vessels enlarged, aorta normal.

Diaphragm. Many haemorrhages, especially over liver area.

Liver. Swollen; marked fatty degeneration. White patches, especially on right side.

Spleen. Enlarged, much congested.

Kidneys. Right much enlarged and congested with cloudy swelling; pelvis filled with urine.

Left same, only slightly less marked.

Cortical vessels enlarged and congested.

Uterus. Much enlarged, not reduced after labour. Many big clots inside. Haemorrhages on surface of uterus and left ovary.

Ovaries, tubes and ligaments congested.

<i>Microscopical examination.</i>	<i>Smears.</i>	<i>Cultures.</i>
<i>Heart</i>	+	+
<i>Lung</i>	+ +	+
<i>Pericardial fluid</i>	+	+

b. The stillborn baby was autopsied on the 23rd of March. Pathological notes taken were as follows:—

FULLTERM FOETUS. No external lesions were noted on the body, skin was pale and showed no petechiae or ecchymoses.

The Placenta was still attached through the umbilical cord to foetus. Both placenta and cord showed no gross changes. Smears from the former showed many *B. pestis*, culture was positive but mixed with other organisms.

Slight caput succedaneum. Some bloody fluid in the subdural space. Films taken from this showed no *B. pestis*, but culture was positive though mixed.

In dissecting out the brain, interior of occiput was found much congested, especially the fontanel.

Brain was congested. Cerebrospinal fluid taken from the fourth ventricle was clear and gave positive results in smears and culture.

The mucous membrane of the *tongue* and *pharynx* was neither swollen nor congested. The tongue showed some haemorrhages at the back near the fauces. The *tonsils* were slightly congested. The *larynx* was markedly congested. There were haemorrhages below the vocal cords, more marked beneath the mucosa of the laryngeal side of the epiglottis.

Trachea and *Bronchi* were of normal appearance. Frothy mucus was present in both main bronchi.

Both *Pleural cavities* contained some non-sanguinous liquid, giving positive plague cultures. *Lungs* had no air in the alveoli and were much congested. Haemorrhages were seen between upper and lower lobes of both lungs as well as on outer aspect of the left lung, where they were arranged in confluent stripes. Culture from the lung was positive.

Peribronchial glands were apparently unchanged.

Some haemorrhages were noted beneath the visceral layer of the *pericardium* also on the mediastinum.

Heart was distended, especially in the right chambers and filled with clots. One large and a few small haemorrhages were seen on the pulmonary valves, beneath the intima of aorta and of pulmonary artery. Smears and cultures taken from both chambers gave positive results.

Liver seemed congested. Small haemorrhages were present in the wall of the gallbladder and beneath the neighbouring liver capsule. Smears and cultures from the liver were positive.

Spleen was firm and congested but not enlarged. Smears from it showed many *B. pestis*, but cultivations remained sterile.

Both Kidneys were congested, and cloudy swelling was not marked; no haemorrhages in the pelvis. Smears taken from both ureters were positive, while the cultures showed plague bacilli mixed with other organisms.

Oesophagus was normal.

Peritoneal cavity contained much bloody fluid, giving positive findings in smears and cultures.

The mucosa of the *Stomach* was slightly swollen and congested, especially near the pylorus. Normal meconium was observed in the large intestine.

Bladder contained urine; its surface vessels were slightly enlarged and the mucosa was somewhat congested.

Uterus seemed normal. Smears and culture taken from its cavity showed plague bacilli.

The results of the histological examination of this unique case will be given under our histological findings.

T A B L E I.
DISTRIBUTION OF PNEUMONIC AREAS IN LOBES OF LUNGS.

No. of P.M.	Age	Sex	Rt. U.L.	Rt. M.L.	Rt. L.L.	Left U.L.	Left L.L.
1	Adult	M	Extensive area	—	Lobular confl.	—	—
2	16	F	Extensive area	Lobular confl.	Lobular confl.	Lobular confl.	Lobular confl.
3	22	M	Patch	—	Lobular confl.	Lobular confl.	Lobular confl.
5	22	F	Lobular confl.	Extensive area	Extensive area	Lobular confl.	Lobular confl.
6	17	F	—	Extensive area	Lobular confl.	—	Central patch
7	8	M	Patch	—	—	—	—
9	31	F	Patch	—	Lobular confl.	—	—
10	35	M	Extensive area	—	—	Lobular confl.	—
11	Adult	M	Central patches	Patches	Lobular confl.	Patches	—
12	10	M	—	—	—	—	Extensive area
13	24	F	Extensive area	—	Central patch	Patches	Lobular confl.
16	24	M	Lobular confl.	—	Patch	Patch	Central patch
18	14	M	—	—	—	—	Lobular confl.
19	24	F	Extensive area	Lobular confl.	Extensive area	—	—
20	5	M	Patch	Early lobular confl.	Lobular confl.	Patch	Patch
21	61	M	—	—	—	Extensive area	—
23	Adult	M	—	Extensive area	Lobular confl.	Patches	Patches
27	24	M	Extensive area	—	—	—	—
32	25	M	—	—	—	—	Patch
4	3	F	—	—	Central patch	—	Central patches
17	1	M	Patch	—	Patch	Lobular confl.	Lobular confl.
22	41	M	Patch	—	—	—	—
28	Adult	M	Lobular confl.	—	Lobular confl.	Lobular confl.	—
31	40	M	Early lobular confl.	—	Early lobular confl.	Lobular confl.	—

TABLE II
LIST OF P. MS. SHOWING CONDITIONS OF LUNGS IN PLAGUE PNEUMONIA.

No. of P.M.	Age	Sex	Date	Marked Pneum. & Pleuritis	Slight Pneum. & Pleuritis	No Pneum. or Pleuritis	Remarks
1	Adult	M	Feb. 27	1	—	—	
2	16	F	Mar. 3	1	—	—	
3	22	M	" 4	1	—	—	
4	3	F	" 8	—	1	—	Child 3 years
5	22	F	" 8	1	—	—	
6	17	F	" 14	1	—	—	
7	8	M	" 18	1	—	—	
8	Stillborn	F	" 23	—	—	1	Stillborn
9	31	F	" 24	1	—	—	Mother of above
10	35	M	" 24	1	—	—	
11	Adult	M	" 25	1	—	—	
12	10	M	" 30	1	—	—	
13	24	F	Apr. 5	1	—	—	
14	2	F	" 5	—	—	1	Child 2 years
16	24	M	" 12	1	—	—	
17	1	M	" 12	—	1	—	Child 1 year
18	14	M	" 19	1	—	—	
19	24	F	" 23	1	—	—	
20	5	M	" 23	1	—	—	
21	61	M	" 24	1	—	—	
22	41	M	" 27	—	1	—	
23	Adult	M	May 1	1	—	—	
27	24	M	" 13	1	—	—	
28	Adult	M	" 13	—	1	—	
29	35	M	" 14	—	—	1	
30	Adult	M	" 16	—	—	1	
31	40	M	" 16	—	1	—	
32	25	M	" 17	1	—	—	Had only 1 patch
33	26	M	" 17	—	—	1	
34	Adult	M	" 17	—	—	1	
35	35	M	" 19	—	—	1	
36	25	M	" 19	—	—	1	
37	30	M	" 20	—	—	1	
39	25	M	" 21	—	—	1	
Total		25M9F		19	5	10	Incl. Stillborn

A STUDY OF THE MORBID HISTOLOGY OF THE 1921 MANCHURIAN PLAGUE EPIDEMIC

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CONTENTS

- | | |
|--|-------------------------------|
| 1. Introduction. | 10. Placenta, Umbilical Cord. |
| 2. Larynx. | 11. Brain. |
| 3. Trachea. | 12. Pancreas. |
| 4. Bronchus. | 13. Suprarenal Gland. |
| 5. Tonsils. | 14. Thymus. |
| 6. Uvula, Tongue. | 15. Kidney. |
| 7. Oesophagus, Stomach. | 16. Heart. |
| 8. Uterus, Ovary, Tubes. | 17. Spleen. |
| 9. Testes. | 18. Liver. |
| 19. Lung, Bronchioli, Pleura. | |
| 20. Lymph Glands (mesenterial, cervical, bronchial). | |
| 21. Discussion and Conclusions. | |

1. *Introduction.*

The following descriptions are based upon the examination of small specimens from twenty three cases of pneumonic plague of the epidemic of 1921 in North Manchuria supplied by Dr. Wu Lien Teh. As it has not been possible to give a detailed description of every case, the following is a summary of the pathological changes found in the different organs as represented by the specimens which have been examined. Slides have been attempted with each specimen to make possible an examination of the histological changes, the elastic fibres, "gitterfaser," fibrin and also the bacteria. The latter are stained by Loeffler's methylene blue, azure II, Giemsa, Romanowsky, etc. For the purpose of control Gram's staining was used.

In view of our studies of the pathological anatomy of pneumonic plague during the epidemic of 1911 in South

Manchuria we have herein incorporated some of the results of the investigations of this disease which were made at that time.

2. *Larynx*. One case.

No severe histological changes, except a considerable hyperaemia in the submucous tissue. Some round-cell infiltration was noted. Both hyperaemia and round cell infiltration occurred in and about the mucous glands.

3. *Trachea*. Seven cases.

In some cases the epithelial cells of the mucous membrane seemed to be but slightly affected, if at all. In others, however, desquamation, necrosis or destruction of epithelial cells were occasionally noted, but without serious ulceration. At the points where the epithelial cells were destroyed plague bacilli were found in the surrounding tissue. In one case there was a dense accumulation of bacilli in the sub-epithelial tissue in the neighborhood of a mucous gland. The changes in the mucous membrane varied in different cases and in location. Without exception, however, a marked hyperaemia of the submucous tissue was noted, and this was often accompanied by round-cell infiltration. The outer walls appeared to remain unaffected and to show no evidence of hyperaemia or cell infiltration.

4. *Bronchus*. Five cases.

The condition of the wall is similar to that of the trachea. In addition, small local haemorrhages occurred in the mucous membrane, which penetrated into the subepithelial tissue. The inner surface of the wall was covered with detritus, blood, or desquamated and destroyed epithelial cells. A high degree of hyperaemia was noted in the submucous tissue, as well as some round-cell infiltration. Blood capillaries in the mucous glands were dilated and a mucous condition marked many of the gland cells. Plague bacilli were abundant in the mucous membrane and submucous tissue. In some cases these even penetrated as far as the mucous glands. Where the augmentation of the bacilli in the mucous membrane was very dense, the covering epithelial cells were destroyed. The histological changes noted in these specimens are found to be less marked in the cases that were studied in the plague of 1911, but the general conditions are very much the same.

5. *Tonsils*. Eight cases.

Marked hyperaemia in all cases. The Tonsils were more or less rich in parenchymatous cells. In some cases the so-called "germinating centers" appeared greatly enlarged. These

"germinating centers" were composed of large mononuclear light cells, including histiocytes which act as phagocytes. These histiocytes containing plague bacilli were also frequently observed in other parts of the glands. It is noteworthy that in some cases these enlarged "germinating centers" were not to be found.

The epithelial layer covering the surface of the tonsil appeared in most parts to be generally intact, or at least not destroyed. Here and there, however, destruction of epithelium took place, accompanied by destruction or necrosis of tonsillar cells of the superficial layer, and an emigration of leucocytes. More or less of inflammatory exudation might have occurred but no considerable destruction of tissue was noted.

Plague bacilli, more or less isolated or in masses, existed in the cavities of the surface and also within the tissue. The penetration of the bacilli into the tissue at the points of epithelial destruction was dense and the bacilli were found to be massed about the lymph follicles. Besides the plague bacilli numerous Gram-positive bacteria were also observed, especially in the cavities of the surface, and occasionally in small numbers within the tissue.

While these tonsils showed considerable hyperaemia and some necrosis of the cells with inflammatory exudation, the pathological changes were not particularly marked. In comparison with the changes that occur in the lungs and neighboring lymph glands they are very slight. This condition agrees with that observed in the epidemic of 1911, with one exception. In that case, the lymph glands of the neck showed such remarkable enlargement with haemorrhages as to indicate that the primary infection had occurred in these glands rather than in the lungs. Also the tonsils and surrounding tissues of the pharynx and larynx suffered great pathological changes. None of the present specimens exhibited any such changes as occurred in this particular case.

6. *Uvula*. Five cases. *Tongue*. Seven cases.

These specimens revealed only very slight pathological changes, although considerable hyperaemia had occurred. The epithelial layer was intact, or at least showed no marked evidence of destruction. In some cases subepithelial round-cell infiltration had taken place.

7. *Oesophagus*. One case.

No special histological changes in the tissue. There was, however, a marked dilatation and swelling of the small veins. The epithelial layer remained intact without evidence of haemorrhage. Within the submucous tissue small colonies of

plague bacilli were noted in the clefts of the tissue which were unaccompanied by inflammatory changes. These colonies of bacilli were probably the result of postmortal propagation.

Stomach. One case.

No marked changes in the tissue of the mucous membrane. The epithelium remained intact and showed no signs of mucous obstruction. The veins and capillaries of the mucous membrane and submucous tissue indicated a condition of hyperaemia. No plague bacilli were found in this specimen, but whether this was due to their absence or to poor staining is not clear.

8. *Uterus, Ovary, Tubes.* Two cases each.

No marked pathological changes. A puerperal condition existed in one case and the tissue of the uterus showed characteristic tissue changes, but there was no evidence of plague infection. Plague bacilli were noted in the blood vessels only and were not numerous.

9. *Testes.* Two cases.

Tissue changes characteristic of the age of the patient were noted without evidence of pathological changes in the parenchyma or interstitium. The formation of spermatozoa did not seem to be materially affected. Because of inability to perfect the staining of the plague bacilli in the blood it was impossible to determine whether they had been able to enter the lumen of the seminal tubule through the apparently intact wall.

10. *Placenta.* One case.

It is not evident that any special structural changes had been caused by the plague infection. No plague bacilli were noted in the blood vessels of the chorion-villus. In some chorion-villi hyaline-necrotic changes were observed, but these were not necessarily pathognomic of plague.

Umbilical Cord. One case.

No special tissue changes; no plague bacilli were stained.

11. *Brain.* Two cases.

Some postmortal and other artificial changes were noted. Where complete fixation had occurred the nerve cells showed no structural changes. The nuclei of most of the cells appeared intact, but the condition of the Nissle's corpuscles could not be ascertained. There was no evidence of cell infiltration, and haemorrhage occurred only slightly in the pia. Plague bacilli were observed in the blood vessels only, being most numerous in those of the meninges.

12. *Pancreas*. One case.

Plague bacilli only in small numbers in the blood vessels. No marked changes in the tissue of the parenchyma, interstitium or in Langerhans's islands.

13. *Suprarenal Gland*. Two cases.

No structural changes were observed. No marked changes in the "Gitterfaser." Hyperaemia occurred in one case. The cells of the cortical layer suffered no necrotic changes. Contents of lipoid not marked. A slight haemorrhage in the zona reticulus was noted in one case. The medullary layer was well preserved, no destruction or hypertrophy being apparent.

14. *Thymus*. Five cases.

No special structural changes. Plague bacilli were observed in large numbers in a few cases in the blood vessels and capillaries. Isolated bacilli might have been scattered about within the tissue, but no accumulations were found. No necrosis, haemorrhage or inflammatory areas were observed. Hyperaemia evident in most cases.

15. *Kidney*. Ten cases.

Hyperaemia to some extent in every case. While the exact condition of some specimens could not be determined because of postmortal changes, in those which had been properly fixed some cloudy swelling of the parenchyma was noted. Though the specimens were not suitable for fat coloration, numerous fine red granules or droplets stained by Sudan III were found, mainly in the epithelium of the convoluted tubules and also in the limbs of Henle's loop-tubules. While the presence of fat granules does not necessarily indicate degeneration of cells, parenchyma cells which undergo cloudy swelling tend toward slight fatty degeneration. No extensive necrosis, fatty degeneration or destruction of parenchym cells was noted, although in a very few cells a lack of staining of nuclei was evident. The lumina of the tubules in some cases contained hyaline globular substance. The presence of a very small number of hyaline cylinders was noted in a few cases.

The fixation of the tissue did not allow for a satisfactory staining of Altmann's granules. These seemed to be quite irregular and diffused. This change occurs in case of plague, and while it is impossible to reach an exact conclusion on this point in connection with these specimens, it is probable that this condition exists.

Important changes were noted in the glomeruli; hyaline thickening of the capillary walls of the glomeruli was often

evident. This condition was also often observed in the cases during the plague of 1911. While it has not been possible to discover any well marked hyaline fibrous thrombi in the capillaries of the glomeruli, as have been mentioned by some authorities, this does not indicate that they are absent here.

In no case was haemorrhage into the cavity of the Bowman's capsules or the urinary tubules observed. This agrees with the results of investigations made in the epidemic of 1911. Such haemorrhage, if it occurs, must be rather unusual. Only in one case was any cloudy substance noted in the cavity of Bowman's capsules. Also in one other case there was observed somewhat of an accumulation of polynuclear leucocytes in one glomerulus. On the whole, it is apparent that the glomeruli are affected by plague virus.

Plague bacilli in the blood vessels of the kidney and also in the capillaries of the glomeruli were always seen. This was not the case, however, in the lumen of the tubules. In one of these cases a very few isolated Gram-negative bacteria were noted, the morphology of which appeared similar to that of plague bacilli, but it was not possible to identify them as such. In the epidemic of 1911 bacilli were found within the rectus tubules in only one case. While the passage of plague bacilli into the urinary tubules may not be impossible, this is not in any sense a usual or necessary feature. The relatively small number of plague bacilli in the blood vessels of the kidney as compared with those in the liver corresponds to our observations in the epidemic of 1911.

16. *Heart.* Five cases.

In no case was there any marked change in the structure and arrangement of the muscular fibres. In general the form and staining of the nuclei indicated that they were intact. On the whole the striation of the muscular fibres was distinct, but in some cases this showed signs of being disturbed. No marked degenerative changes were observed. Because the fixation of the specimens was not suitable for fat staining, Sudan III did not produce good results. In specimens from the epidemic of 1911 Sudan III revealed fine fatty globules in the muscular fibres. However, so far as these specimens are concerned, the muscular fibres in places were somewhat marked by cloudy swelling and it is possible that they might have undergone some fatty degeneration. No special cell infiltration in the interstitium, and no evidence of haemorrhage. His's fascicules remained unchanged. Because of the method of fixation the glycogen in these fascicules could not be stained. Plague bacilli were always present both in the large blood vessels and in the capillaries.

17. *Spleen.* Thirteen cases.

More or less evidence of hyperaemia, the intensity of which varied in different parts of the organ. There were no indications of marked haemorrhage.

In general the pulp showed no marked cell proliferation which in some cases was more or less hyperplastic. Eosinophile leucocytes and plasma cells were present only in small numbers. A very few giant cells of bone marrow were noted in one or two cases. Evidence of histiocytes or endothelial cells was often noted in the presence of comparatively large numbers of mononuclear cells containing much protoplasm. In some cases relatively numerous polynuclear leucocytes appeared, but in others they were not abnormally present. This condition was also noted in the epidemic of 1911.

No extensive necrosis of tissue of cells was apparent, except in one case, which exhibited a notable anaemic infarct of the spleen. Necrosis of small numbers of cells was observed here and there, however, especially in those localities where plague bacilli were densely accumulated. Often some structureless dirty hyaline substance was noted within the pulp. Such hyaline substance was located between the cells of the pulp or apparently along the reticulum.

The lymph follicles of the spleen were generally small in size, sometimes abnormally so. None showed evidence of marked enlargement. This condition agrees also with investigations made in the epidemic of 1911. The follicles, which consist of lymphocytes, almost always showed clearly defined boundaries, though some exceptions were noted. While the tissue of most follicles remained apparently intact, in some cases there were marked changes. Some deposition of hyaline substance within the follicle along the reticulum between the cells was noted. In one case the wall of the small artery within a follicle had undergone necrosis and in the neighboring area such hyaline deposition between the cells was apparent. In a few cases so called "germinating centers" containing enlarged and lightly stained cells were noted. Some of these were certainly histiocytes formed from reticulo-endothelial cells and acted as phagocytes for the disposal of broken-down cell substance. Some measure of hyperaemia surrounded the follicles.

Deposition of subendothelial hyaline substance was noted in the walls of the small arteries. In one case the "Gitterfaser" of the spleen was stained but this showed no evidence of special thickening or diminution.

Plague bacilli were found to be abundant in the blood space of the pulp. Between the pulp cells they appeared either singly

or in colonies. They were mostly free between the cells but a few were phagocyted. Regarding the phagocytosis of plague bacilli in the spleen the specimens were not suitable for minute examination, and this report must accordingly remain incomplete. But the histological examination of the epidemic of 1911 showed phagocytes originating from endothelial or reticulo-endothelial cells or histiocytes in the pulp. The same condition must have happened in these specimens also.

The lymph follicles were found to contain plague bacilli, but they were less numerous than in the pulp. The bacilli were mainly limited to the surrounding or peripheral zones of the follicles, as very few were noted in the central parts. Gram-positive bacteria in the pulp were found only in small numbers in some cases.

18. *Liver*. Fifteen cases,

The structure of the acini and cellular trabeculum seemed to be intact. No marked dissociation of the parenchyma tissue was apparent. More or less hyperaemia was evident. Some dilatation and fulness of the vena centralis and blood capillaries in the central parts of the acini were noted. The parenchyma cells, on the whole, appeared intact, although there was some evidence of cloudy swelling. The specimens were not suitable for fat staining, but Sudan III revealed in most cases more or less presence of fat droplets. The localization of these fat droplets could not be exactly determined, but in a number of cases these were most numerous in the central parts of the acini.

In general no marked cell necrosis was found, but in some cases the nuclei of the epithelial cells did not respond well to the stain. No inflammatory cell infiltration was anywhere noted; nor were any metastatic suppurative areas, such as those reported by some authorities in the liver in cases of bubonic plague, apparent. Except in the case of a foetus of a plague mother, there was no augmentation of lymphoid cells of lymph follicle-like character, but the presence of these lymphoid nodules might not have been especially abnormal in the foetus.

No signs of haemorrhage were to be found. Plague bacilli were observed in the blood vessels and capillaries in fairly large numbers. Phagocytosis of the bacilli by endothelial cells was taking place. Gram-positive bacteria were found in the blood capillaries in small numbers in only a few cases.

19. *Lung*. Fifteen cases.

Pneumonic plague causes most important and very complicated tissue changes in this organ. Several stages of histological change, from simple hyperaemia, or hyperaemia with slight serous

exudation, to severe cellular exudation, were observed. More or less haemorrhage occurred in various parts of the lungs. Haemorrhage within the alveoli in these cases was not especially severe, but erythrocytes in larger or smaller numbers within the alveoli were always apparent. In most cases, in and under the pleura, haemorrhage was histologically very marked. Some haemorrhage was also noted in the interstitium, especially in the interlobular septum and within the connective tissue surrounding the bronchi and blood vessels. At the root of the lung in the connective tissue surrounding the bronchi and blood vessels severe haemorrhage was sometimes observed.

Fulness and dilatation of the capillaries of the alveolar walls was noted even in portions of the lung where no plague bacilli were found in the alveoli, or no inflammatory exudation had taken place. This hyperaemia was accompanied by serous exudate which filled the alveoli and contained a few exudative cells, especially leucocytes. In the early stages of inflammation, plague bacilli occurred within the serous exudate in fairly large numbers. Where the inflammation was more advanced the alveoli became filled with cells, polynuclear cells being most numerous, but which were accompanied by mononuclear cells, the latter being mostly histiocytes and desquamated epithelial cells. These cells in the exudate were more or less mixed with erythrocytes and serous exudate which contained a comparatively very small quantity of fibrin.

The degree of density of cells in the exudate within the alveoli varied greatly; in some cases the cells were loosely arranged while in others they were closely packed together. In every case the alveoli contained dense masses of plague bacilli. In no other form of pneumonia are the pathogenic bacilli to be found in such abundance. It is also characteristic of plague pneumonia, as compared with ordinary pneumonia, that fibrin exists within the exudate only in small quantities. The other important histological characteristic of plague pneumonia is the frequent appearance of hyaline substance in the alveolar walls. The presence of this hyaline material is accompanied by various changes in the walls of the alveoli. Where these changes occur the latter are irregularly thickened. This enlargement of the alveolar walls in one case was due to a hyaline thickening of the capillary walls, in another it was due apparently to hyaline thrombi within the capillaries, and in still other cases to a hyaline deposition on the outside of the capillary walls or within and inside of the alveoli walls themselves. This hyaline deposition was also observed in the epidemic of 1911. At that time, in addition to a deposition on the alveolar walls, a similar hyaline substance was observed to radiate out from the walls of blood vessels. In the present

specimens a somewhat similar deposition seemed to have occurred, but the radiations were not especially marked.

The character of this hyaline substance is difficult to determine and in chemical composition it may not be identical; a part of it without doubt is composed of fibrin. In one case this hyaline substance within the lumen as well as on the walls of the blood capillaries of a number of alveoli was very clearly stained by Weigert's fibrin-staining method, while the staining of the fibrin itself in the same specimen was not good. It is probable that this hyaline deposition is caused by the severe exudative inflammation of the lung and, excepting the fibrin thrombi, is a coagulated substance resulting from the exudation from the blood vessels. This is not necessarily a specific characteristic of plague pneumonia. The deposition of hyaline in and on the walls of the alveoli also occurs, for instance, in influenza pneumonia. Giant cells of bone marrow are often to be found in the blood capillaries of the lung tissue.

"Gitterfaser" and elastic fibre showed no distinct changes in those portions of the lung which were not affected by inflammatory exudation. Where marked exudation did occur, however, some diminution and even destruction of these fibres were to be noted.

Bronchioli.—Where a small area was affected by plague pneumonia and the inflammation was slight the epithelium layer did not seem to be much affected. It frequently appeared to be regular in form and arrangement. In other cases desquamation and obstruction of the epithelial cells with mucus was present. Within areas affected by an advanced stage of pneumonia, the walls of the small bronchi were more severely affected, inflammatory infiltration of lymphocytes and polynuclear leucocytes occurred both in the walls and in the surrounding tissue, and was always accompanied by hyperaemia. The epithelium was largely desquamated or destroyed. A very interesting and important histological development was often noted in the small branches of the bronchi. Sometimes clusters of bacilli and cells of inflammatory infiltration were proceeding toward the lumen from the outside of the wall, the epithelium apparently still remaining intact. In these cases the destruction of the mucous membrane was being brought about by an invasion of the virus through the wall of the bronchioli rather than from within the lumen itself. On the other hand, at other points in the bronchioli destruction of the epithelium had occurred and small clusters of bacilli were noted in the subepithelial layer without evidence of marked changes in the surrounding wall tissue.

Pleura.—In those areas where the lung tissue showed no hepatisation the pleura appeared only slightly affected. Hyperaemia, however, was almost always seen. Also a thin lining of fibrous substance appeared to cover the surface of this portion of the pleura. The surface cells beneath this lining did not seem to be much affected although necrosis occasionally occurred. The pleura covering a pneumonic area is usually much more severely affected. A more or less fibrous lining with many leucocytes appeared on the surface, while the cells beneath usually suffered destruction or necrosis. The small blood vessels on the inner side of the pleura showed exceptional hyperaemia. Haemorrhage in the pleura was very marked and formed a well defined layer beneath the epithelium. In addition there was often a pronounced infiltration of leucocytes into the subpleural and pleural tissue. Fibrin may also occur in very small quantities in these areas of pleuritis, but it is not especially marked. As will be mentioned later, plague bacilli are densely massed in the subpleural and pleural tissue. Occasionally at points on the pleura rather isolated colonies of plague bacilli accompanied by some leucocytes were noted. These patches did not appear to result from the general process of pneumonic hepatisation.

Investigation of the distribution of bacilli in the lung tissue is most important in studying the pathology of pneumonic plague. They are abundant in the alveoli within areas of hepatisation and also where the alveoli have discharged only serous exudate. In the former case, where the alveoli are filled with cellular exudate, the bacilli are densely massed in the spaces between the cells. Of this great multitude of bacilli only a few seem to be phagocyted by the cells within the exudate. In some cases, the phagocytes for plague bacilli could only with difficulty be observed in the pneumonic area. This condition is also to be noted in the lumen of the bronchi. These cells which do act as phagocytes for the plague bacilli are mainly large mononuclear cells, most of which seem to be histiocytes. To what extent polynuclear leucocytes may act as phagocytes to the plague bacilli is difficult to determine.

While plague bacilli are to be found in abundance throughout pneumonic lung tissue, including the blood vessels and capillaries, the greatest accumulations occur in the lymph vessels and lymph spaces of the tissue. The lymph vessels at the root of the lung and those accompanying the branches of the bronchi and neighboring blood vessels are very often most densely packed with plague bacilli. The pathological change in the walls of the bronchioli, as noted above, where the destructive process was working through from the outside of the wall towards the mucous membrane within, was due to the virus being brought by the lymph vessels along the bronchioli.

It was peculiar of many of the blood vessels within the plague pneumonic area that their walls were surrounded by marked accumulations of bacilli. This may be due to some perivascular space which may provide passage for lymph and thus becomes densely filled with bacilli. Very often the wall of the blood vessel itself is invaded by the bacilli and if this is not too thick, as in the case of the veins, an accumulation of bacilli may penetrate to the interior where the endothelial cells are destroyed. In this way great numbers of plague bacilli enter the blood stream. These changes in the walls of the blood vessels were also noted in the epidemic of 1911. The lymph vessels, greatly dilated and filled with bacilli, contain also polynuclear leucocytes and lymphocytes and some blood.

While the bacilli found in greatest abundance in the pneumonic areas were almost entirely plague bacilli, mixed infection in varying degrees was not uncommon. In most of these specimens, gram-positive bacteria, such as diplococci, streptococci, staphylococci and other bacilli, were noted in the blood vessels and alveoli. The number of these bacteria, however, was much smaller than the plague bacilli. In a few cases the number of these other bacteria was fairly large in the pneumonic area, and they might have aggravated the pneumonic condition of the lung, but the chief source of pneumonic infiltration, whose manifestations were almost identical in all cases, is due to plague bacilli.

20. *Lymph Glands.* Mesenterial two cases, cervical two cases, bronchial five cases.

In the two cases of mesenterial lymph glands hyperplasia of cells accompanied by more or less hyperaemia was noted. In one of these a diffused hyperplasia of lymphoid cells had occurred, while the other case was marked by a proliferation of endothelial cells which had filled up the dilated lymph sinus. So called "germinating centers" in the follicles were not well developed. No necrosis, haemorrhage, or inflammatory areas were observed, and polynuclear leucocytes appeared only in small numbers. Bacilli were not numerous in the blood vessels. Examination of numerous slides revealed no plague bacilli in the tissue of the mesenterial lymph glands. In an occasional slide among the cells of the tissue a few bacilli were noted which resembled plague bacilli, but the exact character of the former could not be determined.

One of the two cervical lymph glands showed marked hyperaemia and proliferation of the endothelial cells of the dilated lymph sinus. In some the follicles "germinating centers" appeared. As to the existence of plague bacilli because of poor

staining, no definite results could be obtained. It is at least certain that no large accumulations of bacilli existed, and no tissue changes characteristic of plague were observed.

The other cervical lymph gland proved to be severely affected by chronic tuberculosis. A fibro-caseous substance occupied almost the whole gland, lymph tissue remaining only in one corner. This lymph tissue, especially within the lymph sinus, contained small accumulations of bacilli which resembled plague bacilli. Whether plague bacilli can penetrate into a tubercular area is an interesting question. So far as this specimen is concerned, no plague bacilli were found in the fibro-caseous substance.

The bronchial lymph glands, being most intimately connected with the lung, are the ones which undergo the severest histological changes in pneumonic plague. The entire tissue of the gland becomes heavily infected with plague bacilli. Where these were abundant, the cells of the gland were diminished in number. Cells undergoing necrosis were seen. No marked blood extravasation within the gland was observed. On the other hand the normal tissue structure of the lymph gland was severely affected. Elastic fibres and "Gitterfaser" were less stained and became attenuated or even more or less destroyed. Augmentation of bacilli occurred more markedly in the peripheral zone than in the central portions, and more in the lymph sinus than in the lymph follicles and medullary fasciculus. The presence of bacilli could be noted within the follicles, where they were found scattered among the lymph cells, particularly if the gland was severely infected, but the lymph follicles were always less affected by the bacilli than other parts of the gland. Often follicles were observed to be surrounded by a layer of varying thickness consisting of a mass of bacilli. These seem to offer more resistance to the effects of plague virus than other parts of the gland tissue, but eventually they undergo a gradual atrophy and destruction.

Infiltration of polynuclear leucocytes into the tissue of the lymph gland occurred to some extent. Sometimes these appeared in fairly large numbers but no suppurative areas were noted. A limited amount of serous infiltration was occasionally observed and staining revealed the presence of fibrin, but only in small quantities. A deposition of hyaline substance on the walls of blood vessels and the reticulum, present in cases during the epidemic of 1911, may also have occurred, but it was not prominent. In spite of the presence of great numbers of bacilli and some augmentation of leucocytes and mononuclear migratory cells, phagocytosis in the lymph glands could not be clearly ascertained. In some cases no cells carrying plague bacilli could be found. But investigations during the epidemic of 1911 in-

licated that phagocytosis in the plague bubo did occur to some extent, especially in the early stages of the infection. This was noted particularly in the lymph sinus. In those cases the swollen and desquamated sinus cells or endothelial cells, which partook of the nature of histiocytes, acted to a large extent as phagocytes. Other mononuclear cells may also have become phagocytes. It was occasionally noted furthermore that blistered phagocytes which were carrying lymphocytes and blood pigment had also absorbed some plague bacilli. Where the augmentation of plague bacilli in the lymph sinus became very marked the swollen and desquamated cells and phagocytes disappeared. This is perhaps the reason why in the present specimens, which show advanced stages of plague bubo, phagocytosis is difficult to determine.

In those areas where carbon dust had been heavily deposited in the bronchial lymph glands the accumulation of plague bacilli was less marked.

The relation of the penetration of plague bacilli into tubercular areas of the bronchial lymph glands is the same as in the cervical gland mentioned above.

The tissue surrounding the bronchial lymph glands in the root of the lung was always infected with dense accumulations of plague bacilli, and severe haemorrhage was often noted. Lymph vessels near the bronchial glands appeared dilated and filled with bacilli. Numerous leucocytes, lymphocytes, and some red blood cells were also found in these lymph vessels.

21. *Discussion and Conclusions.*

The most important tissue changes caused by pneumonic plague occur in the lungs and neighbouring bronchial lymph glands. In these organs the plague bacilli are most abundant and the tissue changes which take place are mainly due to the presence of these bacilli. The lungs are affected by hyperaemia, serous and cellular exudation and more or less haemorrhage. The specimens examined in connection with this report did not, however, reveal specially marked haemorrhage into the alveoli. Fibrin within the alveolar exudate was inconsiderable. Hyaline substance, the origin of which is more or less intimately related to that of fibrin, was in most cases found deposited on the alveolar walls.

These tissue changes, together with the enormous increase of plague bacilli in the pneumonic areas, are characteristic of plague pneumonia.

The plague bacilli are abundant not only in the alveoli but also in the interstitial tissue, especially in the lymph vessels and spaces in the immediate neighborhood of the bronchi and blood

vessels and under the pleura. In these lymph passages the plague bacilli are usually found to be most densely massed. The lymph vessels at the root of the lung in the neighborhood of the infected bronchial lymph glands are likewise always greatly dilated and filled with bacilli. The lymph vessels seem to provide an especially favorable medium for the augmentation of the plague bacilli and at the same time they serve as convenient canals along which the latter can proceed to other parts of the tissue.

Examination of the lung shows small areas of plague pneumonia developing in the peribronchial alveoli. The multiplication of bacilli in the peribronchial and perivascular lymph vessels and spaces is a most important cause of this lobular pneumonic infection. On the other hand, the spreading of pneumonic areas may also be due to the inhalation of plague bacilli from the upper and wider parts of the bronchi and bronchioli into the deeper recesses of the lung. In this case the mucous membrane of the bronchioli is affected from within the lumen and the plague bacilli penetrate from the mucous membrane, whose epithelium is more or less destroyed, into the tissue of the walls. This inhalation of bacilli may also carry them directly into the alveoli.

The peribronchial and broncho-pneumonic areas whilst expanding join with each other, thus diffusing the pneumonic infiltration through a large portion of the lung. When, however, the process of pneumonic infiltration occurs over a wide area in a very short space of time its relation to the condition of the bronchioli is not clear.

As to the condition of the cervical, mesenterial and bronchial lymph glands in pneumonic plague, it is the latter which are by far the most severely affected both as to pathological tissue change and the presence of bacilli which are especially augmented in the lymph sinus. The tissue of the mesenterial lymph glands showed only slight evidence of the presence of plague bacilli, and in the cervical glands no large accumulations of bacilli were found. Plague infection in an individual may result in some cellular hyperplasia in these lymph glands. The tonsils are only slightly affected, comparatively small accumulations of bacilli being found and these mainly in the superficial layer.

Modes of Infection.

During the International Plague Conference which was held in Mukden in 1911, two opinions were presented regarding the mode of plague infection. Those holding one opinion argued that the primary infection occurred in the tonsils and that the lungs were secondarily infected by virus carried by the blood. Others insisted that the virus was directly inhaled into the air passages.

The writer (A.F.) supported the latter position at the Mukden Conference, and this present investigation offers no reasons for altering the opinion then set forth.

In no case in the above examination did the tissue changes in the tonsil or the accumulations of bacilli justify the presumption that this organ could be the seat of primary infection. On the other hand the lungs and neighboring lymph glands are always incomparably more severely affected and more densely infected by the plague bacilli. The tissue changes in the tonsils are more probably due to secondary infection by plague bacilli carried by the sputum. The histological condition of the tonsils as compared with the tissue changes in the bronchus and lungs leaves no doubt but that the primary infection follows upon the inhalation of the plague bacilli into the air passages. This does not mean, however, that the bacilli immediately reach the alveoli with the incoming breath. The inhaled bacilli may be deposited for a time on some portion of the walls of the air passages, for instance at or near the bifurcation of the trachea. The multiplication and penetration of the bacilli may then occur in the walls and surrounding tissue. The lymph vessels will then be invaded and along these the multiplying bacilli will be carried to the neighboring lymph glands and also toward the periphery of the bronchial system. Likewise, sooner or later, they may also be carried into the ductus thoracicus. At the same time the histological condition of the bronchus, as described above, which showed intensive augmentation of plague bacilli in the mucous membrane and submucous tissue, which are in direct contact with the air, indicates the possibility of the bacilli being spread through the bronchioli and alveoli by inhalation.

These are certainly the most important modes of infection. On the other hand, primary infection of the pharynx and tonsils, which may cause a cervical bubo, is, of course, not impossible; such a case was observed in the epidemic of 1911. In that case the tonsils were severely affected.

Without doubt the lungs and neighboring lymph glands are primarily infected by pneumonic plague, but secondary infection of these organs may, of course, occur as in the case of bubonic plague.

Bacteraemia. Bacteraemia is generally observed. The invasion of plague bacilli into the blood stream takes place through the ductus thoracicus which carries lymph from the infected areas into the veins. Also plague bacilli may directly enter the blood stream within the infected area by breaking through the walls of the vessels, especially the veins, about and within which the bacilli may be accumulated. This penetration through

destroyed portions of the walls of the veins is often observed in the lungs.

Parenchymatous Organs. The liver and kidney show some parenchymatous degeneration or cloudy swelling of the parenchyma which may incline towards fatty degeneration. (The specimens were not suitable for examination of fat.) Severe extensive degenerative changes of the cells is not apparent. Passage of plague bacilli into the urine does not usually occur. The glomeruli are more or less affected. The muscle cells of the heart are even less affected.

Spleen. The pulp of the spleen often contains considerable numbers of plague bacilli. The tissue changes of this organ consist of some proliferation of the pulp cells and endothelial cells in the earlier stages of the infection, some of which finally suffer necrosis. Blood circulation is disturbed. There is some inflammatory infiltration with emigration of polynuclear cells.

Digestive Organ. The tissue of these organs is not markedly affected by pneumonic plague.

Hyperaemia. In all organs more or less hyperaemia in small vessels and capillaries is noted and haemorrhage often occurs. The toxin of the virus may have caused vasomotor disturbance.

Bacteria. In nearly all organs plague bacilli are found in the blood vessels. Next to the lungs and lymph glands, these are most numerous in the spleen and liver. Gram-positive bacteria of mixed infection were noted to some extent in many cases, and sometimes were fairly numerous in the pneumonic areas of the lungs. These bacteria were sometimes observed also in other organs, especially in the lymph glands, spleen and even in the blood vessels of the liver, always, however, in relatively much smaller numbers than the plague bacilli.

Phagocytes. Phagocytes are found in the lumen of the alveoli, in the bronchioli and the bronchi of the pneumonic areas of the lung and also in the spleen. Could the lymph glands have been examined in the early stages of the infection numerous phagocytes would probably have been found there as well. The endothelial cells of the liver also act as phagocytes. Most of the phagocytes are mononuclear cells such as histiocytes and endothelial or reticulo-endothelial cells. The phagocytes within the lumen of the alveoli of the lungs have been generally regarded as desquamated epithelial cells, but the results of many recent examinations of other forms of pneumonia seem to indicate that these have originated from histiocytes or similar cells. To what extent the desquamated epithelial cells may act as phagocytes can only be determined by further study.

Plague Bacilli in the Foetus. Whether plague bacilli can pass over from the plague infected mother through the placenta

to the foetus remains a question of interest. In the one case which has been examined no tissue changes characteristic of plague were found, nor did the stain reveal any plague bacilli in the tissue. It is stated by those who performed the post-mortems in 1921, however, that the cultivation examinations in this case showed positive results.

Comparisons. A comparison of these specimens with the pathologic-anatomic characteristics of the epidemic of 1911 is difficult to make, because pathological changes vary considerably in the same epidemic in different cases, and are also affected by several etiological conditions. A satisfactory comparison could be made after the examination of a great many cases. Because the foregoing examination covers only a limited number of specimens it does not permit of definite comparative conclusions. However, it may be safely said that the pathological changes in the two epidemics are very similar in nature, as is the mode of development of the disease.

THE ORIGINAL HOME OF PLAGUE

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In discussing this problem, two roads could be chosen to reach our goal. One method is the *historical*, by which the history of plague from the earliest times may be studied and traced to one or more common sources. The other method is the *geographical*, by which certain localities known as endemic foci may be collected and criticised so as to ascertain how far they are really independent of one another.

1. GEOGRAPHICAL EVIDENCE

The geographical evidence is first taken. We start with the endemic centres not as they are known to us nowadays, but, wherever possible, as they were mentioned in the pandemic starting in 1894. This is done because new localities have been turned into "endemic foci" since then by the introduction of extraneous infection, e.g. South Africa, California, Java, Senegal, etc. In other words, the distinction between primary, autochthonous or indigeneous plague foci and secondary plague foci has to be borne in mind. That this is not always easy, the following pages will show, but it is the most satisfactory for the purposes in view.

A good beginning may be made with primary endemic foci known about the beginning of the present century. These are:—

A. *In Africa.*

1. Benghazi (Tripolitania).—2. Central Africa (near the Victoria Nyanza.)

B. *In Asia.*

3. Assyr (Arabia).—4. Western Asia with centre in Kurdistan.—5. Kumaon and Gurwhal (N.W. India).—6. Part of Yunnan Province (China).—7. Transbaikalia and outer Mongolia.—8. Inner Mongolia.—9. Thibet.—10. Turkestan.—11. Persia.—12. Astrakhan and adjoining territories.

A. Endemic Plague Foci in Africa

1. Benghazi is the ancient Cyrenaica in the province of Tripolitania, North Africa. Plague was epidemic in Tripoli from 1856-1859 and again in 1874; no definite origin of these outbreaks was traced. There were not only epidemics in the 19th century up to 1843, but the district seems to have been already affected at the time of the "Black Death," when the disease spread from Egypt in a westerly direction along the African coast. Previous to this already there was an outbreak in 1270 in the adjacent Tunis among the army of Louis XI. To return to the 19th century, there was another outbreak in Benghazi 1892. A Medical Commission was sent out from Malta to investigate the disease. It is true that this commission considered the outbreak as one of "spotted fever," but as the cases had "boils and even axillary abscesses," the affection was suspected to be plague. Moreover the appearance of the outbreak coincided with the arrival of a number of Bedouins from the interior. These people—about 20,000—were driven from their homes through famine. It is true, that a pre-existing infection might have gained new impetus among these poverty stricken people, who were crowded together, but no evidence was forthcoming in this respect. "As the disease is also endemic in Central Africa, it could obviously travel under circumstances northwards as far as Tripoli." This opinion was endorsed by Koch and by Bourges.

No further outbreaks were recorded in Tripolitania until 1913 when some cases were observed at Benghazi and in the villages adjoining it. Its plague character was officially denied, but nevertheless there seems to have been no doubt about it. The disease appeared exclusively among the poorest natives and especially among the Soudanese blacks. No definite source of the outbreak was given, but it appeared that Benghazi formed the starting point for overland caravans to Egypt. Testi mentioned an outbreak of plague at Benghazi in the second half of 1913 which was not completely suppressed until the middle of 1915. He thinks the disease imported from the interior and kept up by continued fresh importations from there, stating that the local conditions are against the spread of the disease to any great extent by rats. In 1913 plague was observed at other places of Tripolitania as well, e.g. Derna and Tripoli. The disease appeared again in 1914 at different places, in 1916 in Tripoli among the Italian troops, in 1917 at and near Benghazi. Mazzone who described the 1917 outbreak, stated that there was no previous epizootic, and infection seems to have been introduced by human agency.

2. *Central Africa*: In 1897 Koch and Zupitza discovered an endemic focus in Kisiba, in the then German East Africa,

between the Kagera-Nile and the Victoria Nyanza. Zupitza visiting the spot observed that plague existed not only among the wild natives but also among rats infesting the dark banana growths. This plague focus is apparently of very old age. A long series of outbreaks up to the present year can be traced back to this endemic centre. While stating this we do not want to convey the impression that they originated at Kisiba, because it seems that plague is widely endemic in the Eastern parts of Central Africa. As is quite usual when discussing the history of plague, the German authorities claimed that the infection came from the English territory and vice-versa. "The truth probably is that the endemic area of plague on the western side of the Victoria Nyanza...includes a portion of the German as well as the British territory plague being endemic in both." Furthermore it must be stated that while we owe to Koch and his collaborator the scientific establishment of the diagnosis, mention of the ravages of plague had been made by previous observers, e.g. by Lugard in 1893. It would lead us too far to enter into a detailed discussion of this problem which is further complicated by the circumstance that for some of the outbreaks east of the Victoria-Nyanza importation from India is probable or possible. For our purpose suffice it to state that there exists in East Africa a large endemic area of old standing. It is not impossible that the focus in Benghazi is fostered from it, especially in view of the evidence collected by different observers at different times. Regarding the plague focus in Central Africa it is at present difficult to explain its origin. There is no doubt that general considerations lead us to look for the original home somewhere in Asia, which is the first habitation of rodents. It would of course be possible for plague to reach these regions via Egypt, this apparently ancient homestead of the pest. The most probable explanation will perhaps be found by reviewing the two historical pandemics, when the spreading power of the disease was noticed at its height. There is much evidence that during the Black Death, Upper Egypt was attacked. But one should be wary at jumping to this conclusion, as the Sudan, lying between Upper Egypt and East Africa, seems to have been free of the pest from the earliest times up to 1911. On the other hand, the earliest authentic pandemic recorded, namely Justinian's plague, was stated by Evagrius to have started in Aethiopia, a locality not distant from Central Africa. Payne who considers Egypt one of the original homes of plague, states that "possibly, if we could follow the history far enough back, we might find, that the African was a colony of the Asiatic plague." There are still many doubtful points in the history and geography of the pest in Africa which may well deserve comprehensive

study. Thus not only for Tripolitania, but for other regions in north and northwest Africa, an importation from the interior seems possible or even probable. This seems true in the case of Morocco, where the outbreaks in its northern part appear to be connected with the Draout tribe who live in the Southwest of that country but migrate regularly northwards. It might be interesting to search for some connection between these supposed plague districts and the endemic areas in East Africa.

It is difficult to trace the mystery of African plague without some mention of the ravages in the islands of Reunion and Mauritius. An excellent survey of this problem is given in Bruce Low's report for 1898-1901. A disease called by the name "Lymphangite infecteuse" had existed in Reunion since 1864 or even earlier and was supposed to have been imported in 1864 from Bombay. When Thiroux announced true bubonic plague on the island in 1894, it was at once investigated if the cases occurring since 1864 were not that disease also. This same suspicion was raised in the case of Mauritius, where during 1866-1867 the "lymphangite infecteuse" also raged in epidemic form and killed 40,000 persons.

Egypt: Though visited long ago by plague which spread to other countries (the first authenticated pandemic, Justinian's plague, is said to have started from Pelusium) Egypt does not seem to be a real endemic centre. Simpson points out, that when Egypt was cut off politically and commercially from the East, no plague appeared there and emphasizes that after the introduction of quarantine measures in 1831 the pest soon disappeared. This view is also maintained by other authors, e.g. Kolle. Thus it seems that plague appeared in Egypt only when introduced from outside, lingering often for considerable lengths of time and conveying the impression of endemicity. The disease was probably introduced from the East. Nevertheless it is not easy to trace definitely even the 1899 outbreak whether it originated from India, the Far East or the nearer shores of Arabia. Anyhow no evidence is procurable to show that any of the Egyptian outbreaks arose from the endemic centre in East Africa.

B. *Endemic Foci in Asia*

3. *Assyr:* Western Arabia, especially its high plateau, is generally considered as an endemic focus. Epidemics have been known in this region since 1815, but as Kremer points out, an outbreak was already recorded there in 1157. This was probably not a solitary or indigenous one, as plague was very active and widespread at that time. It seems that the invasion of 1815 was not restricted to Assyr alone, but travelled along the coast and was present in Jambo, Jeddah and up to Mecca.

The endemic focus in Assyria is important because caravan routes to Mecca pass through it, as also others from Southern and Western Arabia.

Nothing is on record of epizootics prevailing among rats and other rodents in this area but it must be added that the evidence is also scanty for human outbreaks.

No records of Assyria were obtainable after 1906, although outbreaks were still reported at Jeddah, etc.

4. *Western Asia*: This "large area, comprising Persian Kurdistan and adjacent parts of Persia, Turkish Kurdistan, and parts of Irak or Mesopotamia on the banks of the Tigris and Euphrates, including Baghdad" can be considered as a whole. Tholozan had already pointed out, that the real endemic centre of this region was to be found in the mountains of Kurdistan. This statement has not been contradicted.

This endemic centre is doubtless one of the most important ones. It has been active up to this day and the oldest records of plague can be traced back to it. If one believes the outbreak mentioned in the Bible to be plague, then it would seem that the affection, starting during the war between the Israelites and Philistines and apparently connected with the "mice of the field" had started from the endemic focus in Western Asia. This might also be true of the plague of Justinian (A.D. 542). Pelusium which had considerable commercial intercourse with these regions could then be looked upon as a distributing centre.

Martin states that there are no records of epizootics prevailing in this endemic area. There seems however no doubt that rats abound in the rice fields of Mesopotamia, and Avicenna (A.D. 908-1037) also recognised its connection with epizootics.

It is known to students of plague history that former authors endeavoured to differentiate between two varieties of plague "Western Asiatic" and "Indo-Chinese." These authors maintain that the two sets of endemic centres are geographically independent of each other, and also that there are marked distinctions regarding their epidemiology and symptomatology. There is however really no difference between the two "strains" of bacilli, and it can be further shown that the two endemic foci, although far remote from each other, are epidemiologically connected. This can be demonstrated by first describing the endemic foci in the Far East and then turning to the connecting links.

5. *Kumaon and Gurwhal*: An account of plague in this region situated in the Northwest of India on the slopes of the

Himalayas, is given in the Local Government Board Report and in the Report of the German Plague Commission. This focus received much attention at one time, when it was seriously considered as the original focus of the outbreak in Bombay (1896).

The first cases recorded were in 1823, but various observers (Hutcheson, Hirsch, Plank) maintained that the disease is of much older standing and "has existed in all probability in the hill tracts of India and China from time immemorial."

This locality is highly situated and sparsely populated, most of the inhabitants living 3000-6000 feet above the sea level; they are poor and dwell promiscuously with their cattle. The physical character of the districts is such that the conditions for a spread of the disease are rather unfavourable; furthermore the population is well acquainted with the disease and flies in time of danger from the affected villages into the forests. The only thing which may seem likely from a theoretical point of view is the existence of a few places within the districts visited by pilgrims, but this is of little practical importance. It is true that the 1823 outbreak was said to have started in the holy places (Kadarnath or Hurdwar), but on the other hand other observers have noted very few infections among visiting pilgrims. Besides, plague was rarely traced from there into the plains of India: only three outbreaks, one (1825-28) in Housi (Delhi Province), one (1836-38) in Bareilly (Rohilcand Province) and one (1853) in the district Moradabad have been known to start from this centre. Different theories were formed for the origin of plague in these districts. It is maintained that the disease "is imported from time to time from Thibet into Kumaon and Gurwhal and is not, strictly speaking, endemic in these districts." The German Plague Commission was rather sceptical about this theory, stating that there is nothing on which it can be based; and pointed to the fact that the region is separated by the Himalayas from Thibet, whose passes are traversable for four months only in summer. Yet the possibility of Thibet being a focus has to be seriously considered. The German Plague Commission seemed rather inclined to trace the cause of the endemicity to the wild rodents in Kumaon and Gurwhal (*Arctomys*, *Leggada Jerdoni*). Regarding the domestic rodents, Planck obtained (up to 1877) out of 40 affected villages only 8 positive findings of infected rats. He described the rats seen by himself as "a more delicate looking grey species." Hutcheson comes to the conclusion that "spontaneous outbreaks are frequently associated with and sometimes preceded by a great mortality among rats, mice, and other rodents."

6. *Yunnan*: It would be rash to consider the whole of this mountainous province situated in the southwest corner of China as endemic for plague. For instance Vadon states that he has not seen a single plague case in Yunnan-fou during four years. Vallet also denies that the plague ever occurs there. In spite of these statements we believe that Yunnan plays an important part in the epidemiology of the disease. Outbreaks have been mentioned in this province for a long time. Minakata quotes as authority the Chinese explorer Hung Liang-Kih (1736-1809) who described plague among rats and human beings in that region. The old age of plague in these parts is confirmed by other observers. A picturesque though not quite accurate account is given in Simpson's book. Rocher, who visited the province in 1871 and afterwards, found that plague had been known there since 1840, but that *long before that time it had existed in the western part of the province without prevailing epidemically*. Rocher mentions the possibility of infection by caravans or pilgrims from Burmah. It seems doubtful, if his theory can be upheld, since we are unable to obtain records of early cases in Burmah. Yunnan is connected by caravan routes with Thibet and it is more probable for plague to have a permanent home in sparsely populated mountain regions than in populated plains. It appears that Rocher himself had doubts about Yunnan being a primary plague focus, saying that the disease was brought there from the mountainous regions west of Yunnan, its central and eastern parts having been affected since the Mohammedan Revolution of 1853. The capital city is said to have been attacked for the first time in 1872-73.

7. *Transbaikalia and Outer Mongolia*: Only an outline of the plague in these areas will be given here, as the course of the disease in these parts is fairly well known through our researches.

The first mention of a disease among the tarabagans and spreading from them to human beings, was made by a layman, Tsherkasoff, in his book "Memories of a hunter in Eastern Siberia 1856-63." He writes: "There are years, in which the natives don't eat the tarabagans, because the latter have an epidemic disease, they die like flies and many incautious natives, satiating themselves with infected tarabagans, not rarely pay with their lives." The first reports made by medical men in this respect are those of Bjeliavski and Rjeshetnikoff. Although these authors and many after them did not see sick tarabagans, they encountered human cases, but the bacteriological proof was wanting. The first bacteriological examinations in man were not made until 1905. It seems that the oldest human outbreak on record occurred in 1863 at Zagan-Oluevski (Transbaikalia), though other cases had evidently happened much earlier in those

regions. Many authors consider that plague in Transbaikalia is of very old age. A good survey of this question for instance is given by Wassilewski. He points out that the natives have known plague for generations, that they possess a working knowledge of the disease and adopt elaborate precautionary measures for fighting it. It is furthermore mentioned that the tarabagan, its habits and disease are part and parcel of the folklore of the Buriats and Mongols and that the disease among these animals and man is recorded in old Thibetan sacred books.

Among the numerous outbreaks, two small ones are on record where the disease started in the present territory of North Manchuria, namely 11 cases (1905) in the districts of the "Solons," one case (1923) near Jakoshih, both former Mongolian districts. In these instances the human outbreak was probably preceded by an epizootic among tarabagans, whose *bootans* (burrows) are plentiful around these parts.

The endemic focus in Outer Mongolia comprises a very large area. This can be shown by two extreme instances. (1) An epidemic occurring in 1899, 450 versts south-east of Urga. (2) A smaller one recorded in 1899, 200 versts east of Kobdo. The distance separating these two points is about 1050 versts (700 E. miles).

8. *Inner Mongolia.* Zabolotny and other Russian observers believe that the district of Weichang (lat. 42 long. 118), the famous imperial hunting park of N. China, is a true endemic centre of plague.

Dudchenko, who had devoted much time to the study of the tarabagan problem in Siberia, considered that the hibernating habits of the animal helped to limit the spread of the disease, inasmuch as the sick ones usually stayed outside their burrows to die. In trying to assign a reason for the almost yearly appearance of the epizootic among tarabagans, he laid stress upon the regular introduction of plague by the pilgrims passing through Weichang. It is true that Catholic missionaries reported cases of bubonic plague at Weichang as early as 1888, and Zabolotny on his visit there in 1898 bacteriologically confirmed the disease. But since that time nothing has been heard, and Chinese medical officers stationed in the neighborhood have not reported any cases for nearly 25 years.

In regard to the 1917-18 (Shansi) epidemic a Russian observer declared the presence in August 1917 of a "winter sickness" which occurs periodically in the Ordos country and in Inner Mongolia.

9. *Thibet.* Another starting point of the Shansi epidemic (1917) may be Thibet. Parry states that the invasion started

in a monastery called Mai-Uh and was carried afterwards to Taochow, Kansu (China). Although he failed to reach the place, he believed that the disease began in a man *after skinning and eating a tarabagan found dead ON THE HILLS*: He adds that these animals are very numerous in the parts visited by the epidemic. This was stated as well by former observers. Skrgivane said that Preshevalski found in Northern Thibet a variety of the marmot called *Arctomys robustus*. According to the Thibetans this animal is also found in southern Thibet including Lhassa. Skrgivane adds the disease is well known in Thibet and it is due to the measures taken by the inhabitants that it does not assume an epidemic form. He reports also that in the Chinese province Kansu, on the northern slopes of the Tian-Shan mountains both epizootics and human outbreaks are seen. This confirms the above statements of Parry. The importance of Thibet had already been emphasized by observers, e.g. (a) Rennie who believed Thibet to be the possible source of the Yunnan and Mongolian outbreaks and (b) Koch who pointed to Thibet as the real centre for the Chinese and Indian epidemics. Attention should be drawn to a fatal disease with buboes ("Beulenbildung") occurring from time to time in the valley of the Salwen river arising from Thibet and flowing through Burmah. Mueller who quotes this, adds that these reports point perhaps to the true origin of the Yunnan plague. It is quite natural that we have no complete data for Thibet, which in a medical sense is still a *terra incognita*. These facts prove that there is no further reason to be sceptical about the presence of plague in that centrally situated country, as the German Plague Commission (1900) without sufficient evidence was obliged to be. Zabolotny maintains that further proof of the existence of plague in Thibet is established by Paltshikovski's findings in Chinese Turkestan which confirm what was suspected by him before.

Before we continue to discuss individual endemic centres in Asia it would be well perhaps to look back. We have thus far seen three different sets of plague foci in Asia: (a) Assyr, (b) Western Asia with its centre in Kurdistan and (c) a larger area in Eastern Asia, which may be considered as one whole.

We are not able to find from available literature any connection between (a) and (b) and, as far as can be seen the endemic area in Assyr (Arabia) stands isolated by itself. Still the fact that this territory is connected by caravan routes with the centre of gravitation of the Moslem faith in Mecca, makes one suspect that plague was at some early period introduced into this country and had there gained a firm and lasting foothold.

We have to show now what connecting links lie between the Western and Eastern areas in Asia.

10. *Turkestan.* As just mentioned we have proof of the former existence of plague in this part of Central Asia. According to the Local Government Board Report, plague was recorded in 1902 from four points in this region (a) Barumsal in the province of Kashgar; (b) Kandshut in the same province; (c) Badaschan on the south western slope of the Pamirs and (d) Schaschpal. Paltshikovski was sent to Barumsal and established the diagnosis of plague bacteriologically. The English report adds "it is believed that plague was brought by travellers from India across the Karakoram Range and the Hindu Kush." We have no further evidence to confirm or disprove this scanty bit of information, but must refer in addition to Zabolotny's statement regarding a connection of this plague area with Thibet and to the existence of the pest in western (Russian) Turkestan. A suspicious epidemic was reported in 1907 from the Atbaschinsk district, on the Aksai plateau. This region is situated in the south east of Russian Turkestan quite near the frontier of Chinese Turkestan. The origin of this outbreak is curious and as follows:—A Kirghese caught a *black marmot*, brought it in his jurte (tent) and skinned it. He fell sick soon afterwards and a localised outbreak of pneumonic plague affecting 46 persons arose, confirmed bacteriologically by Shendrikovski. Another outbreak was recorded in July 1910 in the province of Semiretchinsk and in two villages of the Abbastin quarter in the Prjevalsk district of that province. The last locality lies still farther east near the frontier of Chinese Turkestan.

In the western part of Turkestan plague outbreaks have been known longer. Klodnitzki reported one outbreak in Afghanistan (1884) one at Merv (1885-87), two extensive ones in Astrabad, Meshed (1887) and finally one in Anzob (1898). He traced all these outbreaks to Khorassan. Plague appeared in September 1892 at Askabad. In 1896 there was an epidemic farther east in Merv, suspected to be plague. In 1898 finally there was the well known outbreak in Anzob in the Hissar range south-east of Samarkand (167 versts). The Russian Government asserted that the infection was "imported to Anzob through Baluchistan and Afghanistan from India by pilgrims who had returned from the pilgrimage to Mecca by way of Karachi, a port which was known to be infected. But no facts in support of this assertion have been brought forward." The report adds that it is not unlikely "that in the villages like Anzob in that district *there may be an endemic infection like the 'Mahamari' of Damaun(?) and Gurwhal, which now and again comes in observation in the remote mountain villages.* Levin in a vivid description of the

Anzob outbreak, appeared to regard the disease as endemic and laid much stress upon 30 odd cases of scars which he found in the inguinal regions of persons supposed to have recovered. In some instances these examined traced their sickness to 20 years previously. We think this point worth serious consideration particularly as the locality lies about half-way between the infected areas in West Turkestan and the established endemic area in Kashgar.

11. *Persia.* This country is perhaps not an endemic centre in the strict sense but nevertheless deserves our closest attention. It has been seen already that the outbreaks in the western parts of Persia can be traced to the common endemic centre in Kurdistan. But there are two more groups of outbreaks to be considered.

- (a) Those occurring since 1876-77 in the province of Khorassan. Mahe "*regards this repeated appearance of plague in the N. E. of Persia as pointing to a possible relation between the plague centres of Mesopotamia and Khurdistan on the one hand, and those to the N. E. of the Himalayas on the other.*" Proust also expressed the opinion that *app. the whole of the high-land from the Caspian Sea to the Himalayas* had never been free from plague outbreaks. All the evidence collected by us points certainly in the same direction.
- (b) In 1905 plague was reported from the province of Seistan, starting S. E. of Lake Helmund. Some tried to establish a connection between this and an infectious disease among cattle, which however was finally diagnosed as anthrax. Others thought of an importation from India through merchandise. A third theory was that "the malady had been prevalent among the nomadic tribes for a considerable time before it was recognised as plague and that the precise source of its origin could not be traced." A fourth theory was that plague infection was brought from Astrakhan by water fowls devouring plague sick rats and harbouring their fleas temporarily. Only the second and particularly the third causes seem to us possible; as for the third, we must emphasize the geographical position of this locality in relation to the plague areas in Russian Turkestan. It must be mentioned that no epizootic among rats was found, although fleas abound. In Khorassan, Grekoff succeeded in 1912 to prove an epizootic among field rats.

12. *Astrakhan and adjoining territories.* Attention had been rivetted upon this area since the well known outbreak in

Vetlianka (1878-79). There is however no doubt that plague had occurred in these parts for a long time. A good survey of this question is given in the Report of the Russian Plague Commission. Outbreaks of "pestilence" were already recorded at the end of the 11th, in the 12th and 13th century, but they cannot be considered definitely as true plague. The first authenticated outbreak is that occurring at the time of the "Black Death" (1364). In the following centuries Astrakhan was repeatedly visited by plague, which according to the unanimous opinion of Russian authors was up to the 19th. century always imported into the country and not of an indigenous nature. They believed it was introduced from the west, the only exception being the deadly epidemic of 1692|93. The origin of this visitation, killing over 10,000 out of 16,000 inhabitants in Astrakhan, is not clear, but it seems to have come from the Eastern foci.

While stating that in the 19th. century the Astrakhan District became itself a plague focus, Russian observers seem rather inclined to connect the first outbreak of 1806-08 with the Caucasus, where pest had reigned from 1798 to 1828. Isaëff emphasized that the second outbreak in 1878-79 at Vetlianka was not imported but really endemic. He drew attention to cases of *pestis minor* occurring before the Vetlianka epidemic in the city of Astrakhan (1877) and pointed to a possible connection between these and the fatal plague in Resht (Persia, 1877).

It would be beyond the scope of this paper to enter into a detailed description of the long controversy *pro* and *contra* the endemicity of plague in those regions. Only a short outline of the problem can be given here. For a long time Russian observers contended that plague in those regions was imported and blamed the pilgrims, here as elsewhere, unnecessarily. The camel was once suspected as a carrier of infection, but although its role was suspicious in a few limited outbreaks, this theory cannot be satisfactorily maintained.

The possible role of the hamster was considered too, but as far as we can see, no results were obtained, although a Commission was appointed to investigate this question. Finally it was ascertained, that the actual carriers of plague here are two species of rodents (*Spermophilus* (Suslik) and *Jerboa*). Lately a third rodent, the wild mouse, was found to suffer also from plague epizootics and to be responsible for the epidemics in autumn and winter.

Klodnitzki, referring to the endemic character of plague in the Kirghiz steppes, says that it is impossible to state when plague was introduced, whether at the time of the Vetlianka

epidemic or even earlier. Equally difficult, it seems to us, is the question from where the disease originated—the Western or the Eastern focus as Astrakhan and the adjoining affected territories lie, like Persia and Russian Turkestan, just between the two foci.

CONCLUSIONS

It was the custom of former observers to fix certain definite localities as endemic foci of plague. As will have been seen from our review, it is difficult to draw any sharp line as to where one focus begins and another ends. So far as Asia is concerned, we may safely say that the whole of the central plateau is one huge endemic area. In the north we have recorded outbreaks in Transbaikalia; in the south at Kumaon and Gurwhal; in the east at Weichang and some parts of Inner Mongolia; in the west Kurdistan. The several outbreaks originating in so called endemic foci, as claimed by various authors, are in our opinion only localised manifestations from one common source. In Central Asia the virus is constantly kept alive among the various species of susceptible rodents, which as we have seen suffer from periodical epizootics. Such visitations in their turn may result at one time or another in human cases.

The task of assigning the original role to any particular species of rodents is not easy especially as the information regarding them has not been uniformly worked out. Starting with the oldest known epidemics on record, such as that mentioned in the Bible, one may be tempted to lay the blame entirely upon Western Asia. It is questionable how far one is justified in considering this outbreak as really the first in history. Knowing as intimately as we do the habits of the tarabagan (Siberian marmot) and its close relationship to plague, we may perhaps be excused in assigning to it a principal role in its causation. It is quite possible that if other rodents, especially those marmot-like ones of Western Himalayas, etc., be similarly investigated, they may be found to play an equally important role, and thus help to confirm our view that the original home of the pest lies in that vast central Asian plateau, inhabited by these burrowing animals.

This idea has been held by observers early in the present pandemic, like Le Dantec, who said among other things that the tarabagan was the real cause of plague, and that it, not the rat, should be exterminated.

2. HISTORICAL EVIDENCE

Turning now to the historical aspect of our subject, we find considerable evidence to support the above view. Let us discuss first the famous Black Death of the 14th. century. The

history of its origin, in spite of the voluminous attention devoted to it, is still obscure. Simpson says: "the Russian records place it in India; Grecian in Scythia; the English in the country east of the Indians and Turks; the Arabians in the states of the great Khan of Tartary and in the land of darkness, and the Italians in Cathay (China)...."

One fact stands prominent, namely, that besides the near Orient, Europe and Africa, there were extensive invasions of Asia, particularly China. For instance, Payne states that "in the years 1333-1347 terrible inundations produced famine in which four million men are said to have perished, and at least one great pestilence, credited with a mortality of five millions is recorded."....Other countries in Asia were also visited by the Black Death. Gabriel de Mussis who himself saw the ravages of plague in Caffé (Crimea?) said, "Thus were the orientals in all parts, both those who lived on the south shore and those on the north, struck down by this pestilential disease and almost all of them died. So great was the mortality, that Arabs, Saracens and Greeks throughout the whole of the East gave themselves up to clamour...."

The attention of contemporary writers was drawn to China, not so much because it was the starting point of the great pandemic, but because of its wholesale death toll in that populous empire. For the above reasons, we believe that the Black Death originated not in China but in Inner Asia, that "Land of Darkness" of Arabian historians and that it spread from there eastwards to India and the Celestial Empire, as well as westwards to Europe.

It is not our object to give a detailed analysis of the period intervening between the Black Death and the pandemic which started in 1894. Two points may nevertheless be accentuated to show that even during this comparatively quiet interval there was a tendency for new waves to appear as if starting from one common focus. Thus:

(1) The epidemic in Cutch, Kathiawar (India) occurred in 1812-21 at a time when the pest was equally prevalent in the Levant, spreading to the Lower Danube, Asia Minor, Armenia and Northern Africa, and lasting for nearly 20 years.

(2) The Pali plague (India) of 1836-38 corresponded in time with a fresh and comparatively limited activity in the Levant affecting Turkish Dominions and Egypt. In Rajputana the invasion disappeared at the same time as in the Levant.

In reviewing the pandemic which began in 1894 and was distributed mainly through the busy seaport of Hongkong, we find again plenty of evidence to show its tendency to radiate from a central focus: For instance—

- (1) In 1899 the disease was unusually extensive and virulent in Outer Mongolia, being recorded in three separate regions and killing over 400 persons. The pneumonic type predominated.
- (2) Plague was seen to assume a more active form in Astrakhan about the same time that it invaded India in 1896.
- (3) In September 1899 the Russian authorities at St. Petersburg were alarmed "at the increasing ravages of a form of 'Malarial fever,' spreading over a large area in certain parts of Central Asia. Plague had on some previous occasions been designated elsewhere by the name of epidemic malarial fever."

3. CONCLUDING REMARKS

In conclusion I may say at once that I am not trying to bring forward any new theory. The material for this article has been largely gathered from the work of past authors. At the same time I cannot help feeling that recent workers have been too much engrossed in certain limited foci without paying sufficient attention to their mutual relation to one another. The role of domestic rodents in the epidemiology of plague has been confirmed more than once, but this is the first occasion on which the part played by the tarabagan has been thoroughly worked out. It is possible also that the original virus may rest in the wild rather than in domestic rodents. In the same way, that Central Asia is now generally regarded as the cradle of the human race, as it assuredly is the first habitation of the wild rodents, so we may say, that the original home of plague was also situated in these regions.

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SUMMARY OF PLAGUE EXPERIMENTS ON THE LICE OF TARABAGANS

BY H. M. JETTMAR

It is proposed that a summary of the results of extensive plague experiments on the louse of the tarabagan be set down here. The detailed article has been sent to Germany and will be published in the scientific journals of that country.

Only brief statements and conclusions will be found in the following paragraphs, but because the data might throw some light on the whole plague and tarabagan problem, it was thought worth while to record them in these pages.

1. The louse of the tarabagan (*Linognathoides spec.*) can suck the blood of man, the ground squirrel and the guinea-pig, but will not live on a strange host for any length of time.

4 *experiments*: Ten lice, collected from a healthy tarabagan, were starved for 1-5 days and were put on the fore-arm of a man. A large percentage of them drew blood. The electric light did not disturb the act of biting which lasted half an hour. The bite of the lice did not cause much itching, but a female volunteer exposed to their bite said the sensation was disagreeable. It was observed that the lice often voided faeces in the act of biting.

Tarabagan lice, placed on healthy guinea-pigs, entirely disappeared after a few weeks.

2. When the host dies the louse of the tarabagan quickly passes on to another host.

1 *experiment*: Lice were put on a dying guinea-pig. After its death, the lice had all left and gone over to a living guinea-pig kept in the same cage.

3. Plague bacilli when sucked into the stomach of the tarabagan louse grow rapidly in clusters and finally fill up the lumen of the stomach.

Observation: Four hours after the ingestion of the plague infected blood, the growth of the bacilli could be observed. The accumulation of the plague bacilli clusters was to be seen in the central part of the stomach where the blood was probably in stagnation, while in the periphery the same organisms

grow in a diffused manner, due probably to the disturbing peristalsis of the stomach wall.

Phagocytosis was seldom seen. The bacilli showed bipolar-staining and no involution forms were encountered.

4. Two or three days after the ingestion of plague blood, the tarabagan louse dies of infection, while healthy lice may live up to 10 days or longer when kept on a few hairs in test tubes and at room temperature.

Experiment: As the result of numerous experiments, one can state with certainty that all lice die after once ingesting plague-infected blood. The so-called self-cleaning was never observed. However, investigations with plague infected lice biting healthy or immunized tarabagans were not carried out.

5. The dead plague infected lice are distinguished by a dark red colour, and in transmitted light by a light red colour.

Observation: Microscopically, dark brown sediments of haemoglobin were seen in all parts of the body as well as in the intestinal tract. The trachea was of the same colour and was most distinct, so that the finest ramifications were clearly seen.

In the intestinal tract, clusters of virulent plague bacilli were found in pure culture.

No plague bacilli were ever discovered in other parts of the body .

6. The faeces of the lice contain plague bacilli in pure culture.

Observation: The bacilli were tightly packed in large clusters so that only at the periphery were bipolar-stained bacilli seen at all distinctly.

7. The alimentary canal of the healthy louse is nearly always sterile.

Observation: In the histological investigation, serial sections of over 100 partly healthy and partly plague-infected lice were made, but only on one occasion was a small lump of slender fusiform bacilli (40 microns in diameter) seen in the stomach of an infected louse.

8. Cocciform micro-organisms are found in the ovarian ducts of both healthy and plague-infected lice of the tarabagan, but never in the intestines. These micro-organisms are not pathogenic to guinea-pigs.

2 experiments: Pure cultures of these cocci were easily obtained as follows:—

8 lice, collected from healthy tarabagans, were washed successively with 10% acetic acid, 10% sodium carbonate, and thoroughly in six dishes of sterile normal saline. They were then ground up and the resulting juice was inoculated on agar. Guinea-pigs were infected with a large quantity of these germs in pure culture, but they did not fall sick.

9. After the death of the infected lice, the plague bacilli in the intestines do not show any involution forms until about the 5th day.

Observation: Before the 5th day, the plague bacilli were highly virulent, and it was only after that time that the virulence diminished and involution forms were observed.

10. Plague-infected lice collected from the dead tarabagan were seen to be infectious at least 13 days afterwards, though they were not protected from light or desiccation.

Experiment: The lice collected after the death of the plague infected animal were kept in Petri dishes. At fixed periods emulsions of 5-30 ground-up lice were injected either subcutaneously or intracutaneously into guinea-pigs:—

Time of injection after collecting the lice from dead host in days	Number of lice emulsified	Time of death of guinea-pigs in days	
		Subcutaneous	Intracutaneous
1	5	3	6
3	5	4	5
13	20	12	4½
25	30	alive	alive

11. By placing 40 lice collected 24 hours previously from a dead plague-infected tarabagan on a healthy sisel (*Spermophilus Eversmanni*, Brandt), one can infect it with plague.

1 experiment: The sisel succumbed to bubonic plague 150 hours after the lice were put on its neck. Plague bacilli were found in its bloody nasal mucus two days before death.

The faeces contained highly virulent Plague bacilli.

12. Ten tarabagan lice, whose mouth organs are smeared with a virulent agar culture of plague bacilli, remained healthy.

13. Whilst probably all experiments of plague infected lice undergo changes described in paragraph 5, only one out of many experiments carried out with *B. anthracis* showed similar phenomena. In the alimentary canal of this louse, the anthrax bacilli were seen to have multiplied considerably.

14. The results of the observations outlined in Paras. 9 and 10 are in accord with those obtained by McCoy with the louse of *Citellus beecheyi*.

A SYSTEMATIC EXPERIMENTAL STUDY OF THE PATHOLOGY OF PNEUMONIC PLAGUE IN THE TARABAGAN AND SISEL (SUSLIK)

BY WU LIEN-TEH

AND

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TABLE OF CONTENTS

- I. INTRODUCTION.
- II. TECHNIQUE.
 - a. Preparing the emulsion.
 - b. Inhalation.
 - c. Killing.
 - d. Bacteriological examination.
 - e. Histology.
- III. DESCRIPTION OF EXPERIMENTS (*Table I*)
- IV. GROUP A. Animals dying spontaneously of plague.
 - a. Macroscopical findings (*Tables II and III*).
 - b. Microscopical findings.
- V. GROUP B. Animals killed at varying intervals.
 - a. Macroscopical findings (*Table IV*)
 - b. Microscopical findings.
- VI. Discussion, Summary and Conclusions.
- VII. TABLES I-IV.
- VIII. BIBLIOGRAPHY.

I. INTRODUCTION

Experimental plague infection of arctomyidae through the respiratory tract has been performed several times (Strong 32, 33, Tshurilina and Nossina 34, Tsurumi 35, 36.) The most exhaustive experiments in this connection were performed by Wu and Eberson (40) and by Wu, Chun and Pollitzer (39). The animals were inoculated either by injection direct into the trachea or by spraying. When infected by these methods they developed either septicemic or better *pulmonary* plague (Strong 1 case, Wu and Eberson 4 cases, Wu, Chun and Pollitzer 3 cases) or they succumbed to Pneumonia (Strong 1 case, Tsurumi

4 cases, Wu and Eberson 9 cases, Wu, Chun and Pollitzer 3 cases).

Though in most of the above instances post mortems were performed, no systematic studies of the histological changes were carried out. In order to complete our observations, a fuller series of experiments have been undertaken during the past two years and the results compiled in this report.

Our present observations fall under two groups:

- A. Those upon inhaled animals which succumbed spontaneously to plague or were killed in the later stages of illness when showing distinct clinical signs.
- B. Those on animals killed at varying intervals after inhalation before any marked symptoms appeared.

For our purposes, the animals used were partly tarabagans (*arctomys bobac*) and partly sisels—small grey rodents living widely on the Sungari plain (average weight of 200 grms.). These sisels are highly susceptible to experimental plague infection, the mortality being 100%.

II. TECHNIQUE

(a) *Preparing the Emulsion.*

Emulsions of the lungs from plague infected animals were used. At the beginning of our experiments a series of guinea pigs were infected by lung puncture* so as to obtain a highly virulent emulsion.

The first tarabagan in the series received inhalation of an emulsion of the hepatized lung of the last guinea pig infected by lung puncture. Thereafter parts of the lungs from inhaled tarabagans and sisels were successively used for preparing the emulsions. Approximately 1 c.c. of the organ was cut up into small pieces and shaken in 10 c.c. of sterile saline solution, until a pink emulsion was obtained. This liquid was then poured into a Paroleine atomiser (Burroughs Wellcome & Co.) similar to that used by Martini (21, 22) in his inhalation experiments. About 1—2 hours are usually required for preparing and spraying the emulsion.

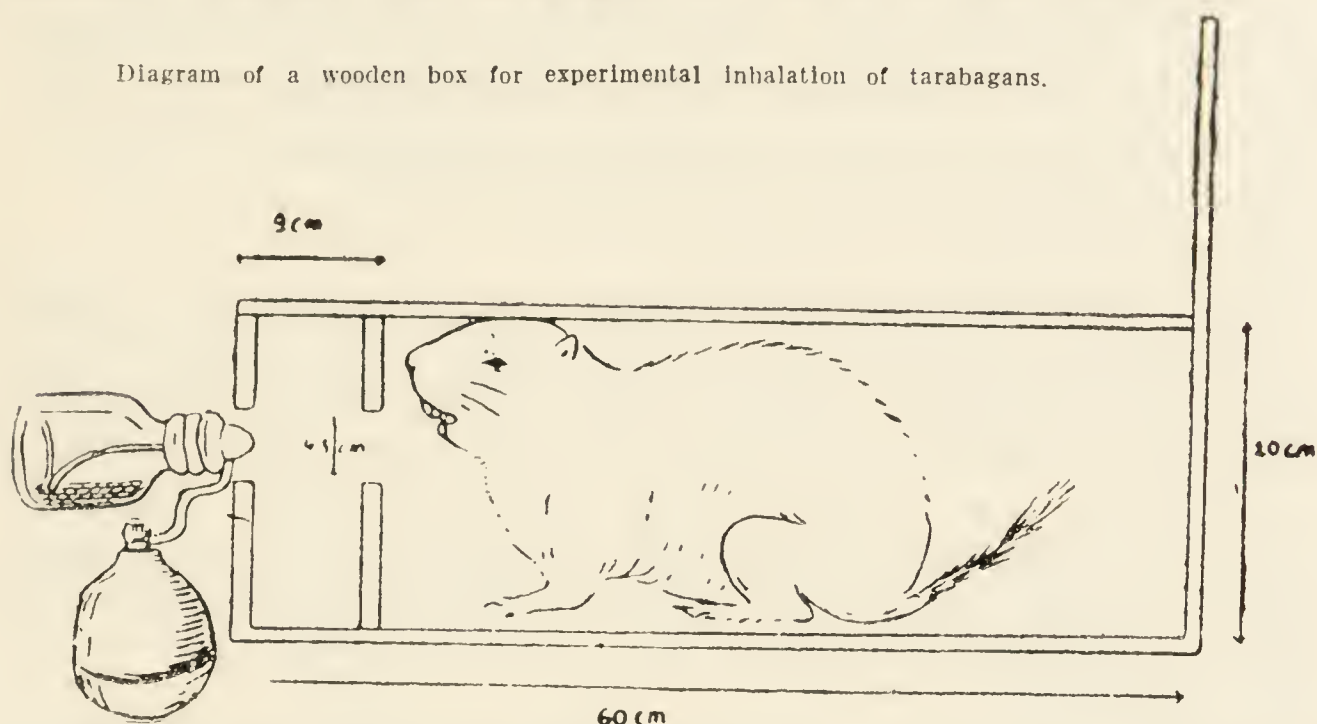
(b) *Inhalation.*

We have considerably improved upon our previous methods. A specially-made wooden box is now used with a partition separating a small antechamber and the main compartment. The latter just accommodated one tarabagan. In the case of sisels, small rat cages were used and placed in the middle of the main compartment. The partition wall had an

* The technique was generally similar to that of Shibayama (28) with one modification: The inside wire of a needle from a hypodermic syringe was dipped into the infecting material (lung) and then drawn back into the needle. The point of the needle was next cleaned with alcohol, and passed through the flame. Finally the needle was inserted into the lower part of the left lung and the thin wire pushed forward. Afterwards the wire was pulled back, and the needle removed. In this way infection of the cutaneous and subcutaneous tissue was avoided.

aperture situated at the level of the animal's head, just large enough for inserting the muzzle of a tarabagan or sisel. Facing this aperture is another slit in the outer wall of the antechamber through which the nozzle of the atomiser was introduced. The antechamber, prevents big drops of the spray from reaching the animal. Further the tarabagan is unable to bite the nozzle.

Diagram of a wooden box for experimental inhalation of tarabagans.



Before spraying, the animal is stirred up by slight thrusts with a blunt stick until it yelps. Then the spray is directed towards it. This moment is chosen, because forceful inspiration promotes the distribution of the spray into the smallest ramifications of the lower respiratory channel.

Early in the series the animals received inhalation for some time—up to one hour—and almost the whole of the emulsion was used. But it soon became evident that a very short period was sufficient. Finally only 40 compressions of the bulb were given, the manipulation lasting one minute, and less than one c.c. of the emulsion being used. After the spray the animal was kept for 5-10 minutes longer in the box so as to allow it to inhale the finest droplets suspended in the air.* All the tarabagans of group B were thus treated for only one minute.

After inhalation each animal was placed in a separate cage. Unfortunately many of the sisels, whether inoculated or not, fell into a dormant state, and succumbed soon afterwards. When killed, intestinal bacteria were frequently found in their internal organs, especially the liver. This kind of necrobiosis has been observed already by Schurupoff (26, 27) who experimented on the *Spermophilus guttatus* of Astrakhan. Mereshkovsky (23) had a similar experience with the Bessarabian sisel, and Jettmar (17) with the *Spermophilus dahuricus* from Transbaikalia. On

* Observations such as those of Lange and collaborators (19, 20) on white mice show that principally the finest droplets, suspended in air, lead to primary infection of the lung. According to Buchner, Martini, B. Lange and others, an atomiser may produce droplets with a diameter of 2-8 microns. Such droplets remain suspended in the air up to six hours (Gotschlich 9).

the other hand, the Mukden marmot (*Sp. Mongolicus*), *Spermophilus Eversmani* from Transbaikalia (16) and the California Ground Squirrel (*Citellus Beecheyi* 7) proved suitable for laboratory purposes.

(c) *Killing.*

The following method was adopted: The animal was held by a pair of padded tongs. Then the skull was perforated by a sharp hook. The animal died almost immediately and no disturbing hæmorrhages into the respiratory tract occurred. The post mortem was performed immediately afterwards.

(d) *Bacteriological examination.*

The following technique was used to discover any bacteraemia in the earliest stages of the disease: blood was removed with a thick canula from the ventricle of the heart, while still slightly palpitating in most of the cases. 2—8 c.c. heartblood, obtained in this way, were divided among several test tubes containing bouillon or agar and incubated for several days or even weeks. Tests on guinea pigs were used in every case to identify the growing colonies.

(e) *Histology.*

For histological investigations pieces of the internal organs of every animal were fixed for a short time in formalin. The respiratory organs of the animals killed in the incubation period were preserved *in toto* in formalin. Then the lung was cut into pieces, every one of which was registered before embedding in paraffin, sketches of the shape being made. The embedded pieces were cut in the following way:

The first sections of each block were carefully investigated. If pathologic changes were seen, serial sections were made of the whole piece. If not, 100-200 microns of the pieces were sliced off and the new surfaces were again investigated, and so on. Thus each piece could be completely searched for morbid features.

The upper respiratory tract was systematically examined in every case; the tonsils received particular attention.

Kossel's method of staining was adopted for the sections:

10 c.c. conc. aq. sol. Meth. Blue (Hoechst) is diluted in 100 c.c. distilled water.

To this, add 30 drops of a 5% sol. of Sod. Carbonate (cryst.). 6 c.c. of 1% aqueous Eosin A extra (Hoechst) is now gently added while shaking.

Formation of sedimentation must be prevented.

Solution must be prepared and filtered immediately before use.

Stain section in this solution for 10-30 m. Differentiate in dil. acetic acid (1 drop glacial in petri dishful of dist. water) until pink colour appears in parts.

Pass through 95, then absolute, alcohol until no more blue comes out. Pass through Xylol and mount in cedar wood oil.

By this method even under low magnification, the blue-violet coloured granules of the basophile cells frequently present in the lymphatic tissue of the tarabagan, could be differentiated from the dark-blue and bipolar plague bacilli. We proceed now to a:—

III. DESCRIPTION OF THE EXPERIMENTS

Table I summarises the general results (Group A).

The above shows a mortality of 100% corresponding to the experiments performed in tarabagans by Wu, Chun and Pollitzer (39) who found all their inhaled animals succumbing to plague. As may be seen from the table and the findings mentioned later on, the length of exposure seems to have no influence either on the duration of the illness or on the character of the pathologic changes.

The long duration of the illness in the tarabagans No. 2, 6, 9, 12, and 13 is somewhat significant in connection with the primary pneumonic changes found. Similarly individual resistance seems to affect the results. Thus, the bigger among the tarabagans succumbed to the primary pneumonic form, while the smaller ones took a pulmonary aspect.

38% of the tarabagans (5 out of 13 animals) suffered from primary pneumonic plague; the percentage among the sisels was approximately the same.

Every animal with clinical symptoms, even in the initial stage, showed bacteraemia. Smears and cultures were made from the following organs of every tarabagan:

1. mucus in trachea.
2. lung.
3. heart blood.
4. liver.
5. spleen.
6. gall bladder.

In every case positive bacteriological results were obtained from the lungs, blood, liver and spleen of the tarabagan. The smears of the mucus and gall bladder sometimes proved negative or doubtful, being often contaminated.

Smears from heart, liver, and kidneys of the sisels were mostly positive.

Table II supplies details of the macroscopical lesions found in the organs of plague inhaled animals (Group A.)

The histological lesions in the inhaled animals are as follows:
Cervical Glands.

These are never severely affected. Tarabagans 3 and 10, sisels 5 and 2 showed a moderate swelling in the submaxillary lymph glands. In sisel 2 hyperaemia was noted in addition.

Fauces showed no marked lesions. Occasionally a few small haemorrhages were seen at the root of the tongue. Only once (Tarabagan 9) were larger haemorrhages noted in the mucous membrane of the nose.

Tonsils. In most of the cases they were not swollen but had a pale colour, with no haemorrhages. Big clusters of plague bacilli were often seen in the blood vessels. Here and there they abounded in the network of the lymphatic capillaries with increase of the polynuclear leucocytes. Occasionally small groups of plague bacilli were observed wandering through the intact epithelium of the crypts; they were never found in the lacunae.

Tarabagan 6 showed severe lesions, such as, large haemorrhages, infiltration of leucocytes, and masses of plague bacilli which had almost destroyed the normal structure of the tonsil. The haemorrhages extended at some places even to the surrounding connective tissue. Necroses were not observed in the tonsils, though they abounded in a tracheobronchial lymph gland, thus showing the primary lesion to be present in this gland and not in the tonsils.

Trachea. In almost every case, marked tracheitis was present. Large haemorrhages are common in the posterior wall of the trachea, especially in tarabagans. Frequently they pervade the whole substance. In some cases they extend only into the serosa. Large haemorrhages are often found at the bifurcation. The contents of the trachea consist of a sanguineous froth, containing blood corpuscles, desquamated epithelial cells, a few leucocytes, and masses of bacteria. A gelatinous, frequently haemorrhagic oedema, between trachea and oesophagus and in the mediastinum, was found in every tarabagan and sisel dead of pulmonary plague (except Tarb. I and Sisel I).

Albrecht & Ghon (1) consider this kind of oedema as typical of the primary bubo. Accordingly the glands enclosed in the oedema may be regarded as the primary bubo in inhalation plague.

Tracheal and Tracheo-bronchial Lymphatic Glands.

The deep tracheal or tracheobronchial glands showed in every case most severe changes. Often they were literally embedded in haemorrhages, and it was quite impossible to distinguish them from their surroundings. The microscopical lesions were not less marked, such as intense destructive haemorrhages, dense infiltration of polynuclear leucocytes and plague bacilli in the tissue of the lymphatic gland and the neighbourhood, and necrotic foci.

The small accumulations of lymphatic tissue around the main bronchi were often entirely invaded by plague bacilli. In such

cases the polynuclear leucocytes numbered more than the lymphocytes.

Bifurcation of Trachea and Main Bronchi.

It has already been mentioned that the bifurcation of the trachea frequently shows severe haemorrhages. As a rule these are found in the posterior wall of the trachea and bronchi where the cartilage is absent. Here they reach beneath the epithelium of the respiratory tract, and surround the big blood vessels. The coat of the veins is practically destroyed, and often shows signs of hyaline degeneration, while the arteries suffer less severe changes. Big, compact masses of bacteria are found in these haemorrhages as well as in the lymph follicles of the posterior wall. Plague bacilli are interspersed everywhere in the haemorrhages, sometimes entering the lumen of the vein. The epithelium of the bronchi is affected likewise by these organisms. Smaller and larger groups of bacilli also invade the submucosa, where they form masses especially around a small blood vessel. The epithelium is loosened at these spots, while the bacillary masses proceed to the lumen of the bronchi. A similar process has been reported by Fujinami & Wu Lien Teh in human plague (8). Sometimes bacilli are found in the basal cells of the epithelium, accompanied by a few leucocytes or lymphocytes. The organisms have apparently emigrated either from the submucosa, or directly from the contents of the bronchus.

Lungs. Here a distinction must be made regarding the type of plague from which the animal suffers. In the case of septicemic or better *pulmonary* plague, the capillaries are filled with plague bacilli, which may outnumber those found in the larger blood vessels. This was especially marked in sisel 9, killed in the dormant state 46 hours after inhalation. In some instances the capillaries are dilated, and contain numerous polynuclear leucocytes. They may even be entirely blocked by bacteria for a long distance. Hyaline degeneration of the walls is found very often in such areas followed not seldom by hyaline obturation of the lumen. (Tarabagan 1, 7, 8, 10, 11, Sisel 2, 4, 6).

The *walls of the alveoli* are thickened, and infiltrated with leucocytes and red corpuscles. Sometimes slight hyaline degeneration is observed, especially beneath the pleura (Tarabagan 8, Sisel 2, 3, 9,). As a rule, these changes are more marked in sisels.

Collapsed *alveoli* are sometimes met with near the big bronchi and blood vessels. The epithelium of the alveoli is much swollen with slight desquamation of the cells. These latter show badly stained nuclei, and vacuolisation of the protoplasm; sometimes they contain blood pigment (sisel 2 and 4). As a rule, the lumen of the alveoli is empty. Some haemorrhage occurs in T I, and sisel

1, 2 and 3. The alveoli under the pleura are filled with a serous exudate, in which many plague bacilli are suspended. Similar changes may be found in subcutaneously infected tarabagans as well, and thus are not characteristic of a pulmonary infection. Such exudate is sometimes observed in other parts of the lungs, usually in alveoli situated near the larger bronchi (Tarb. 7, 10) where plague bacilli agglomerate. The number of desquamated epithelial cells may be increased, but not the leucocytes. In a number of cases the bacilli suspended in the exudate have multiplied so quickly as to fill up the whole lumen of the alveolus. This may be followed by a secondary infiltration of leucocytes. In this way small pneumonic patches develop under the pleura, and also along the bronchi. (Sisel 2, 3, 5. Compare the findings in pneumonic plague).

The Bronchioli (small bronchi) in Pulmonary Plague show the same lesions as the larger branches. A condition of acute inflammation exists surrounded by haemorrhages extending to the smallest ramifications. Compact bacillary masses are found in these haemorrhages. Similar changes are seen around the blood vessels of the lung and may extend as far as the precapillaries (interalveolar). See Tarb. 1, 7, 10, Sisel 2, 4. In other instances the perivascular spaces are considerably dilated, and filled with a granular or filiform exudate, enclosing numerous plague bacilli as well as some leucocytes, and red corpuscles. Wu and Lin (34) found similar changes in naturally infected tarabagans. This infiltration of the plague bacilli around the blood vessels is in some animals very intensive. The *blood vessels* are surrounded by compact masses of bacilli, lying at first in the adventitia and later on infiltrating the walls of the vessels. (Tarb. 1, 3, Sisel 4, 6.). In such cases swelling and proliferation of the endothelial cells are observed. These bacillary clumps are extraordinarily large in Tarb. 7 and 8, where the diameter measures twice that of the blood vessel. The bacillary masses approach the blood vessels as far as the precapillaries. The above changes have not been noted in other organs of the same animals. The lung vessels of tarabagans 7 and 8 contain a comparatively small number of organisms with no bacillar thrombi inside. In tarabagan 1, 5 and 10, also sisel 2 and 4 such thrombi are encountered even in large blood vessels. The *lymph-vessels* are much dilated and blocked with plague bacilli, so that in stained specimens they may appear to the naked eye as prominent dark-blue spots. Haemorrhages may also occur around.

Primary Pneumonic Plague.

Wu & Eberson (40) and Wu, Chun & Pollitzer (39) have reported that the lesions found in the primary pneumonic plague of arctomyidae correspond to those observed in man. The microscopic changes in the animals dealt with in this paper differ like-

wise in no material way from those already described in human pneumonic plague.*

Sisel 7, however, has to be mentioned here. This animal was killed on the third day after inhalation, and showed almost no symptoms of illness. Post mortem revealed 50-100 partly confluent pneumonic patches of different sizes throughout the lung. The smaller ones especially displayed the same features as those already mentioned in cases of pulmonary plague. The alveoli showed numerous leucocytes, plague bacilli and debris. Hyaline degeneration of the alveolar walls was seen. In the capillaries plague germs were rarely detected; in the spleen they were also seen with difficulty.

No haemorrhages with groups of *B. pestis* were observed at the bifurcatio tracheae or in the walls of the larger bronchi.

The following paragraphs apply to both Pulmonary and Primary Pneumonic Plague.

Pleura.

In pulmonary cases with no pneumonic patches the pleura is almost unchanged. Sometimes the epithelial cells are swollen and lifted up by a small haemorrhage. Here and there circumscribed pleurisy and crowds of plague bacilli are seen, invading the pleura from small secondary pneumonic patches lying beneath. In sisel 3 these invasions are very numerous and extensive.

In animals affected with primary pneumonic plague a fibrinous pleurisy is always found over the surface of the inflamed lobes. Small necroses caused by invasion of bacillary clumps from the lungs are frequent in such cases.

Aorta and Blood vessels on the Hilus pulmonum.

The adventitia shows frequently extensive haemorrhages and clumps of bacilli. The media of the larger veins is often torn asunder by haemorrhages, but the walls of the arteries, particularly of the aorta, show only hyperaemia, sometimes slight extravasation, and isolated groups of *B. pestis*.

Heart.

Haemorrhages were often seen in the muscular layer of the heart, leading sometimes to necrosis of the muscular fibres. In other cases haemorrhagic stripes, densely infiltrated with plague bacilli, separate the degenerated muscular fibres from one another. Even where haemorrhages have not occurred the bundles are loosened by the dilated capillaries. In the perivascular spaces of the capillaries crowds of plague bacilli are found, such changes being most marked in Tarb. 7 and 8.

* The microscopic lesions in the pneumonic foci of guinea pigs infected by lung puncture conform in the main to those found in the primary pneumonic foci of the inhaled arctomyidae.

Spleen. This shows in a few cases necrosis, (Tarb. 9, 10). As a rule there is only hyperaemia with all the features of an acute plague tumour.

Liver. In most of the cases acute congestion is seen. The cellular trabeculae are reduced in some areas, while in others abundant fat droplets and blood pigment inside the liver cells are observed. Sometimes small haemorrhages, followed by slight necrosis of the liver cells occur.

Plague bacilli are abundant in the capillaries and haemorrhages. Frequently the cells of the capillary endothelium show phagocytosis, but the bacilli seemed rather to propagate than to perish in them.

Kidney. The common features are hyperaemia, small haemorrhages and slight degeneration of the epithelium. Here and there small necroses were to be seen originating from the haemorrhages or from a capillary vessel, blocked with plague bacilli.

Alimentary Tract. The intestine often shows haemorrhages of various size. The large ones are situated in the serosa, rarely in the submucosa, and harbour numerous clumps of plague bacilli. The villous coat is only slightly changed. In some cases the capillaries are enlarged and filled with plague bacilli, some being encountered in the surroundings. Occasionally the epithelium is slightly desquamated and some red corpuscles or plague bacilli may be seen wandering through its intact layer. Sometimes larger haemorrhages into the lumen are observed. The lymph-follicles show no marked changes, but the lymph-capillaries may be filled with bacilli. In such cases the mesenteric glands are inflamed as well. In no case, however, do they show severe lesions or the characteristic features of a primary bubo.

GROUP B: Inhaled tarabagans, killed at varying intervals after inhalation before appearance of marked symptoms.

Table IV supplies the details of the inhaled animals as well as morbid changes found at P.M.

This table demonstrates macroscopically the gradual development of pathologic changes. These begin with typical gelatinous oedema on the posterior wall of the lower portion of the trachea and marked swelling of the tracheal or tracheo-bronchial lymph glands. Soon afterwards hyperaemia and catarrhal symptoms of the mucous membrane of the trachea may be observed as well as hyperaemia of the lungs and beginning congestion of the spleen.

The tests on guinea pigs with material from different organs and with cultures from the heart blood produced some remarkable results. Thus:

- (a) Although 9 guinea pigs were inoculated with material taken from tarabagan I (killed 24 hours after inhalation), none fell sick. This material was taken from:

- i. The soft and hard palateinoculated intracut.
- ii. The nasal cavity " "
- iii. The contents of duodenum " "
- iv. The blood of the left ventricle " subcutan.
- v. An emulsion of max. lymph. gland . " "
- vi. An emulsion of right tonsil " "
- vii. An emulsion of mesenteric gland ... " "
- viii. An emulsion of right hilus pulmonum " "
- ix. An emulsion of apex of right lung .. " "

(b) Guinea pigs inoculated with material from the trachea of tarabagan 2 (killed 30 hours after inhalation) likewise showed negative results.

(c) A guinea pig, infected subcutaneously with cultures from the heart blood of tarabagan 6 fell ill with plague but *survived for 11 days*, showing extensive suppuration of inguinal buboes and secondary pneumonia. Apparently, the bacilli were considerably weakened. On the other hand, plague bacilli cultivated from tarb. 7 (killed 72 hours after inhalation) proved fully virulent, for they killed guinea pigs promptly in 4-6 days with acute bubonic symptoms.

These facts seem to prove either (a) that the germs had entirely disappeared from the upper respiratory tract, perhaps even from the tested parts of the lung or (b) that the plague bacillus had been weakened by the protective powers of the tarabagan. Thus they were unable to further infect guinea pigs intracutaneously or subcutaneously. The later explanation appears more plausible.

These observations correspond to the results of authors performing inhaling experiments with different kinds of bacteria (Lange, Keschischian, Novosselsky, etc.), who found that germs perish quickly after inhalation or become much weakened temporarily with changes in their antigenic qualities.

Histological Findings in Group B.

The study of the histological lesions found in these killed animals helps to elucidate to a certain degree the development of plague infection in arctomyidae. Tarabagan 1 and 2 (killed 24 and 30 hours after inhalation) showed no histological changes at all, except perhaps slight hyperaemia in the lung capillaries.

The first marked changes were found in *Tarabagan 3* (killed 42 hours after inhalation). The histological features were present exclusively in the parenchyma of the lung; other organs including the upper respiratory tract remaining unchanged. No plague bacilli could be detected in the tonsils, trachea, main bronchi, or mediastinal lymph glands.

The pathological changes in the lungs apparently originated from two small foci, namely:

- (a) *The first focus* consists of a small bronchial lymph gland adjoining a bronchus with diameter of 0.3 mm situated in the lung parenchyma distant approximately 6 mm from the left hilus.

Sections from the centre of this small focus show the following histological details:

The big *bronchus* has an unchanged mucous coat except where the upper-mentioned lymph gland borders. Here the mucosa and submucosa are completely infiltrated with leucocytes and nuclear debris. The normal structure of the tissue has disappeared, so that no boundary can be seen between the mucosa and the lymph gland. The epithelium is swollen, and immediately under it groups of *B. pestis* may be seen.

The lumen of the bronchus however has no pathological contents and plague bacilli are absent.

The lymph node itself is much enlarged and densely infiltrated with leucocytes and debris. Prominent groups of plague bacilli abound in the centre of the gland which is surrounded almost everywhere by haemorrhages partly filling the lumina of the neighbouring alveoli.

The bronchus is accompanied by a big vein, containing many leucocytes in its lumen, while the endothelium undergoes early desquamation. Some leucocytes are seen wandering through the wall of this blood vessel. The lymph spaces in the adventitia are much enlarged and filled with serum containing lymph cells.

The neighbourhood of the focus in the left lung presents mainly changes in the blood vessels and lymphatics, while the respiratory system is almost unaffected. Here and there groups of collapsed alveoli alternate with emphysematous spots; occasionally a condition of hyperaemia is observed. The walls of the bigger blood vessels are frequently loose and interspersed with leucocytes. Often the endothelium is desquamated.

The dilated lymphspaces and lymphatics of the adventitia form not seldom a broad ring around the blood vessel. These rings are characteristic features of the early stage of the disease. The lymph nodules in this area show often proliferation, and are invaded by pus cells. They also harbour numerous pigment cells, but plague bacilli were absent. Here and there the precapillaries are almost blocked by accumulations of polynuclear cells.

- (b) *The second focus* is situated in the centre of the right middle lobe among the alveoli. Though numerous sections were made, no direct connection with any bronchiole could be found.

The focus has a diameter of about 200 microns and consists of a small number of collapsed alveoli surrounded by an emphysematous area. Besides the elements of the alveolar walls there

are in the focus numerous polynuclear leucocytes, nuclear debris and a central dense cluster of plague bacilli. The original structure of the tissue has almost disappeared.

The alveoli situated between this focus and a neighboring bronchus are somewhat collapsed and hyperaemic. Their capillaries and precapillaries contain numerous polynuclear leucocytes.

The bigger blood vessels of this area show enlarged lymphspaces filled with serum, but no increase of lymph cells.

The whole of the right middle lobe is similarly changed with atelectases, hyperaemia of the alveoli, accumulations of polynuclears in the precapillaries, enlarged lymphspaces in the adventitia of bigger blood vessels containing an increased number of white corpuscles.

The other lobes of the lungs proved to be free from the above changes. The haemorrhages in the alveolar tissue beneath the pleura (mentioned in Table IV) display no changes suggesting the development of a plague focus.

In the lungs of the Tarabagan 4 (killed 45 hours after inhalation) plague bacilli could not be identified with certainty. In some parts of the left upper lobe and of a small right median lobe, however, marked histological changes were observed suggesting the invasion of *B. pestis*. In these areas emphysematous spots alternated with collapsed alveoli. The latter showed pathological changes, such as infiltration of alveolar walls, their capillaries and precapillaries by polynuclears, the last being sometimes blocked. At some places, usually in the middle part of a collapsed area, the leucocytic infiltration is so dense that the pressed alveoli form an irregular shaped nodule with destroyed nuclei in the centre. Probably these nodules represent the early stage of such a plague focus as described in tarabagan No. 3. Plague bacilli, however, could so far not be demonstrated in these areas. Some of the bigger blood vessels surrounding such areas contain an apparently increased number of white blood elements.

A bronchus, situated near one of the above mentioned nodules, shows early infiltration of the adventitia with polynuclears as well as numerous eosinophile cells. At one spot the infiltration has already penetrated the mucosa, and early nuclear degeneration is observed. The accompanying vein has enlarged lymph spaces containing fluid and an increased number of lymphocytes.

The remaining parts of the respiratory tract displayed no changes. In the tonsils was seen a condition of hyperaemia in the subepithelial layer, but the epithelium was intact and plague bacilli were absent.

The epithelium of the trachea shows at places permeation of small groups of erythrocytes into the lumen. The outer layer, especially the lymph nodules on the posterior wall, seems un-

changed. The tracheo-bronchial lymph glands are hyperaemic, but not infiltrated with leucocytes or plague bacilli. In both spleen and liver no unusual changes were observed.

Tarabagan 5 (killed 54 hours after inhalation) showed already some plague foci in different parts of the lung. These are right lower lobe, right middle lobe and right median lobe. No changes were seen in the entire left lung or other lobes of the right lung.* The largest accumulation of *B. pestis* was found in some small bronchial lymph glands situated at the bifurcation of two small bronchi. On the whole these foci demonstrate the same histological features as the lymph gland in Tarabagan 3. In some of these, however, the changes noted were more advanced, as testified by thicker crowds of bacilli invading the wall of the adjoining bronchus on a higher scale, and also wandering through the epithelial layer. Likewise they penetrate the infiltrated adventitia and media of the neighbouring veins, but are not found in the intima. In some parts near the affected lymph node small groups of plague bacilli were found in the lumen of the bronchus, mixed with polynuclear leucocytes. Some alveoli in the immediate neighbourhood of the affected lymphglands are collapsed. In a few, desquamated alveolar cells, polynuclears as well as numerous plague bacilli were found. When nuclear decay was present in such areas, it was rarely possible to find the proper limits of the lymphgland.

The lung district belonging to such an affected gland shows enlarged lymph spaces around the blood vessels as far as the precapillaries. These lymphspaces contain a homogeneous fluid, in which an increased number of lymphocytes and polynuclears are suspended mixed with a few plague bacilli. The alveoli of such areas are partly emphysematous and hyperaemic. In some places isolated red corpuscles wander through the walls into the lumen of the alveoli.

The histological changes in the lungs of Tarabagan 6 (killed after 52 hours) differ in no material way from those found in the previous ones.

The main difference lies in the fact that bacteraemia has taken place already. Serial sections of the most affected lymphgland reveal invasion of plague bacilli into a lung vein.

The following histological features are observed:

The lymph gland situated amidst several bronchioles is much enlarged. Masses of polynuclears and nuclear debris have entirely destroyed the original structure of the tissue. This gland communicates by large infiltrated lymph ways with

*The right lung of the Tarabagan has two posterior median lobes attached to the right main bronchus, besides the usual three lobes.

the neighbouring nodules, which are likewise severely affected. The lymphgland is filled with plague bacilli wandering in continuous masses through the loose bronchial epithelium. The lumen of the bronchus contains clumps of plague bacilli mixed with numerous polynuclears.

The whole lymphgland is interspersed and surrounded by haemorrhages.

The walls of a big lung vein, enclosed in the proliferated lymph gland, are loosened and densely infiltrated with leucocytes and red corpuscles. At some spots they are torn asunder including the elastic coat. The intima is much thickened by leucocytic infiltration. The endothelium is swollen and desquamated. In many parts this is absent thus allowing plague bacilli to enter the lumen of the vessel, where single organisms surrounded by endothelial cells and leucocytes are seen.

Following the more advanced process in the lungs, the tracheobronchial lymph glands and the lower tracheal glands are much affected. Some of these are enlarged, showing proliferation of their cells and marked infiltration with decaying polynuclear leucocytes. In some parts many plague bacilli are observed, showing by their pair-like disposition a condition of highly active growth.

The trachea bears signs of a slight catarrh, but the epithelium is intact, and advanced changes in the walls are not observed. The lymphatic tissue on the posterior wall of the lower trachea shows slight proliferation and infiltration by polynuclears. Here plague bacilli in moderate numbers are seen. In the upper part of the trachea no pathological changes are noticed in the lymphatic nodules, nor are plague bacilli encountered.

The tonsils show no unusual changes, and plague bacilli are absent. All other organs are unchanged with the exception of the spleen. Here the characteristics of an early acute plague tumour are evident.

In tarabagan 7 the pathologic changes are still more developed, and similar to those described in advanced pulmonary cases.

Spacious exudate rings around the blood vessels, desquamation of the endothelial layer, severe catarrh in the air passages containing big clusters of plague bacilli, suppuration of the lymphatic nodules with extension of the process to the neighbouring alveoli are the main features.

In the upper respiratory tract marked changes are seen such as have been described already in advanced cases.

VI. DISCUSSION, SUMMARY AND CONCLUSIONS

The histological findings based upon a systematic study of over 4000 sections obtained from 20 tarabagans and 9 sisels tend to show that the *B. pestis* invades the intact wall of the lower respiratory tract, thus causing infection and death of the animals.

Primary buboes were always found among the bronchial and tracheo-bronchial lymph glands—macroscopically characterised by considerable swelling, haemorrhages and a gelatinous sanguineous oedema. The nasopharynx, conjunctiva and alimentary canal never revealed primary lesions nor offered such features as those described by Schurupoff²⁶. This author inoculated plague bacilli either on the conjunctiva or nasal mucous membrane in three series of *Sp. guttatus*, and fed another lot with infected material. He found enlarged submaxillary and postauricular lymphglands with infiltration of the surrounding tissues. Besides, all his sisels inoculated on the mucous membrane showed a bloody secretion from their nostrils containing *B. pestis* in almost pure culture, while those infected orally sometimes developed bloody diarrhoea.

Our inhaled animals did not show any of the above features. Though the tonsils were thoroughly examined in every case, only one pair was affected. But even here there were more marked primary changes in the lungs.

Although the major air-channels, such as, larynx, trachea and main bronchi, showed most severe lesions in advanced cases, primary foci were never observed in them in the early stages of the disease, notwithstanding the fact that the lung tissue had already displayed distinct morbid changes. These findings do not speak for a primary infection of the big air passages.

Of course, most of the inhaled bacilli, especially those suspended in the larger droplets, are retained by the walls of the tracheo-bronchial ramifications. It seems, however, that the organisms are quickly ejected, weakened or killed *in situ*. This view is supported by the fact that the presence of plague bacilli cannot be ascertained either by histological or animal tests until two days have passed after inhalation.

Nevertheless, it must be mentioned that the *secondary* changes in the trachea and main bronchi begin at quite an early period. On one hand, plague bacilli rapidly propagate in the lymph nodules or lymphglands near the large bronchi, and enter the lumen of the latter. On the other, clumps of pus and epithelial cells, containing masses of *B. pestis*, are carried away from the foci of the deeper respiratory tract to the major air-passages along with the products of secretion.

With regard to the *smaller bronchi*, it may be possible for a primary infection to be localised in them. Microscopical findings in Tarab. 5 and 6 (Group B) seem to support this contention. Furthermore, it appears as if the inhaled bacilli had penetrated primarily the mucous membrane of the bronchus adjoining the affected lymphgland. A similar mode of infection was suggested by Birch-Hirschfeld (Deutsches Archiv. f. klin. Med.) and Abrikossow (Virch. Arch. Bd. 178, 1904) for primary localisation of the tubercle bacillus in the lungs. These authors found among other things primary localisation in the mucous membrane of a middle-sized bronchus in the apex of a lung.

The presence of plague bacilli in the different lymph vessels may possibly be caused by congestion and reversion of the lymph stream. Two facts, however, are against this view:

(a) In a still earlier stage (Tarab 3), only the centre of the lymphgland and the region of the different lymphatics are affected, while other parts, especially those bordering the mucous membrane of the bronchus, seem to escape.

(b) The epithelium of the bronchus is affected only in one region corresponding to the centre of the lymphgland. Plague bacilli are not likely to have invaded the gland exactly and exclusively at this point.

Excluding the above mentioned possibilities, we believe that the deeper parts of the respiratory tract, namely, the *branchioli respiratorii* (infundibula) and the alveoli, are the most likely spots for the final entrance of the invading organisms.

It may seem strange that the inhaled droplets carrying plague bacilli can wander directly through the sinuous bronchial ramifications to the alveoli without being arrested by their walls. It has to be remembered, however, that numerous exceedingly small droplets with a diameter of only some microns are produced by the atomiser. Earlier experiments with various bacteria have proved that such droplets may be suspended in the air for several hours, and may also penetrate to the alveoli (Arnold², Buchner,⁶ Hildebrandt,¹³ Nenninger,²⁴ Paul,²⁵). Under such circumstances, Langer and colleagues, Griffith,¹¹ etc., believe that the smallest droplets are responsible for infection in inhaled rodents.

Moreover, it has been ascertained that the undamaged walls of the alveoli may be traversed by bacteria; also that small particles of coal dust, dead bacteria, etc., are able to wander through the intact alveolar epithelium—to be found shortly after inhalation in the alveolar lymph spaces, lymphatics and bronchial lymphglands (Lange and Keschischian¹⁹).

Our findings of animals killed in the early stages indicate that plague infection apparently occurs in the area of the alveoli in two ways:

(a) *More common mode.* The inhaled droplets conveying plague bacilli enter the lymphspaces (Saftkanalsystem) of the alveolar walls through a gap between their epithelial cells and produce the first changes on their way to the bronchial gland, as has been described in Tarab. 5 and 6 (Group B). It is interesting to find the plague bacilli in these lymph spaces rather evenly distributed, and only on rare occasions arranged in small groups. It seems as if the respiratory movement promoting the flow of the lymph stream assists the spread of the bacteria evenly. This method of infection displays no obvious lesion of the alveolar wall at the site of entry.

It has been known for some time that plague bacilli can wander through the intact skin and not produce any local pathological changes. In the same way they are probably capable of passing through the alveolar epithelium.

In 1888, Cornet made similar statements regarding the action of the tubercle bacillus (Wiener Med. Wochenschr.), which were confirmed by other workers, such as, Behring, Calmette, Orth, etc. Thus plague inhalation in arctomyidae at least may likewise be interpreted mainly as a *cryptogeneus infection*.

There is still another analogy between the mode of infection in inhalation plague and tuberculosis. As has been mentioned, the mediastinal lymphglands of Tarab. 4 and 5 of Group B (killed in an early stage of the disease) were markedly enlarged. In T 5 especially these glands could even be *macroscopically* recognised by the encircling gelatinous haemorrhagic oedema. Microscopically, they showed evident proliferation of the lymph tissue and infiltration with polynuclears. No plague bacilli were, however, visible in hundreds of serial sections made.

Similar observations are reported by Herring and MacNaughten (Lancet, 1922, I) when experimenting with *B. tuberc.* subcutaneously upon guineapigs. It seems therefore that in both cases the soluble toxin of the bacilli first enters the lymphglands and prepares them, though ineffectively, for the fight against the incoming organisms. Though there are essential analogies between the mode of infection in inhalation plague and inhalation tuberculosis, an important difference lies in the fact that tubercle bacilli are carried away in the lymph stream by phagocytic cells, while plague bacilli are freely suspended in the fluid of the dilated lymph spaces, exudate cells never enclosing them.*

(b) *Less common mode.* The pathological changes observed here in the alveoli cannot be so satisfactorily explained as in mode a. Some collapsed areas in the lung tissue are infiltrated with many pus cells and contain crowds of plague bacilli. These

*Primary plague pneumonia in arctomyidae shows also no phagocytosis of *B. pestis* by alveolar or dust cells, though sometimes observed in human cases (Strong, 32 Wu and Woodhead, 42 Fujinami and Wu, 8 etc.) and frequently in the early stages of the disease (Jettmar 14).

foci increase in size, lead to hepatisation of other parts of the alveolar tissue, and finally lobular pneumonia.

Two more questions remain to be discussed:

(i) How does hepatisation of lung areas in arctomyidae occur? In primary pneumonic plague this apparently starts in the majority of cases from the above-mentioned alveolar atelectases as a result of primary localisation of the *B. pestis* from the droplets. In other instances they may arise from a severely affected bronchial lymphgland, and then the process involves the neighboring alveoli through emigration of leucocytes and plague bacilli (Tarab. 6 and 7 Group B, and several tarabagans and sisels of Group A).

In advanced pulmonary cases, secondary pneumonic foci may develop under the pleura, as already mentioned under Group A: here the alveoli are filled with a serous fluid in which plague bacilli are suspended.

(ii). How does the septicemia originate?

The lung veins with their thin walls, situated in the immediate neighborhood of an infected lymphatic nodule, are much exposed and evidently allow *B. pestis* at a very early stage of the infection to enter the lumen of the vessel (Tarab 6 Group B). About the same time several lymphatic glands, communicating with the main lymph channels are already attacked; an influx of the infected lymph into the blood stream follows as a sequence.

While trying to apply our knowledge as gathered from those experiments upon tarabagans and sisels, two aspects may be emphasised: I. From our studies of the 1920-21 human outbreak in Manchuria (38, 39) we came to the conclusion that besides manifest pneumonic plague, there was a group of cases designated as *pulmonary* met with mostly towards the end of the epidemic. Other workers have previously observed such cases but have merely called them 'septicemic' without realising perhaps their true significance. Our present experimental observations have, we hope, materially contributed to a knowledge of this type of respiratory infection.

II. In an elaborate report by Kulescha (1915)¹⁸ he proposed, for further support of his theory of tonsillar infection in pneumonic plague, that systematic animal experiments should be made to ascertain the true mode of entry of the infecting organism. Our extensive series of investigations may be said to have fitted in with his desires, but they have produced exactly the opposite results that Kulescha expected. In other words, we are more convinced than ever that infection in plague pneumonia is not a faucial, or even a tracheal, one, but essentially an invasion of the deeper portions of the respiratory tract.

TABLE I

(List of Animals under Group A.)

a. TARABAGANS.

No.	Size:	How long inhaled?	Inhaled with:	Sleeping?	Killed? Died?	Survived after infection	Type plague
1	Big	1 hour	Emuls. lung from gp.	No	Died	under 4 days	Pulm.
2	"	30 min.	" " tarab. I	"	Killed when dying	9 "	Pneum.
3	Small	"	" " "	"	Died	over 3 "	Pulm.
4	Med.	"	" " "	"	"	4 "	"
5	Small	25 "	" " 2	"	"	3 "	"
6	Big	"	" " "	"	Killed when dying	6 "	Pneum.
7	Med.	15 "	" " 6	"	Died	3 "	Pulm.
8	"	"	" " "	"	Killed when dying	3 "	"
9	Big	2 "	" " 7	"	Died	7 "	Pneum.
10	"	1 "	" " 9	"	"	over 3 "	Pulm.
11	Small	"	" " "	"	"	3 "	"
12	Med.	"	" " from gp.	"	"	under 6 "	Pneum.
13	Big	"	" " "	"	"	5 "	"

b. SISELS.

1	250g	5 min.	Cult. lung tarab. II	No	Died	under 4 days	Pulm.
2	220g	"	" " "	"	"	4 "	"
3	210g	"	" " "	"	"	4 "	"
4	250g	"	Emuls. lung sisels 2 & 3	"	Killed when dying	4 "	"
5	220g	2 "	" " "	"	Died	under 4 "	" Pneum. incip.
6	210g	"	" " "	12 hrs.	"	5 "	"
7	210g	"	" " "	No	Killed before any signs of illness	3 "	Pneum.
8	200g	"	" " "	12 hrs.	"	"	"
9	260g	"	Trach. mucus sise j9	24 "	Died	5 "	"
					Killed in dormant state	46 hours	Pulm.

TABLE II

Showing macroscopic changes in inhaled tarabagans.

No.	Path. changes in ext. lymph. glds.	Tonsils & fauces	Trachea;	Tracheal and tr.— bronchial glds.;	Lungs and Pleura;	Spleen.	Other organs;
1.	No	Small haem. in tons. angle	Tracheitis, large haem. at bifurc.		No pneum.; lower lobes cong. Haem. in walls of lung veins.	Acute sw.	Cort. haem. in kidneys.
2.	No	No marked ch.	Tracheitis.	Trach.—br. gds. enl., dark-red.	Numer. confl. pneum. foci of greyish colour, with red periphery, esp. in lower lobes. Fibrin. pleuritis.	Twicenorm	Pericard. with fibrin.-sanguin. exudate. Adhesions.
3.	One submax. lymph-gl. sw.	Small haem. on tons.	Tracheitis. In cent. Trach. port. and bifurc. glds. hyper- extens. haem. Haem. aem., sw., gland on oedema bet. oes. and arc. aortae hyper- trach. aem. and sw.		All lobes cont. air; sections covered with haem. froth.	Small, pale.	Haem. in walls of arcus aortae; liver enl. Mesent. glds. dark- red. Haem. in wall of alim. tract.
4.	No	No marked ch.	Tracheitis. In cent. Embed- port. and bifurc. dif- rages. fuse haem. Haem. oedema bet. oes. and trach.	Embedded in haemor- rhages.	All lobes cont. air; no pathol. changes.	1 ½ enl.	Mesent. glds. enl., dark-red
5.	No	No marked ch.	Spotted haem. in cent. Glds. enl., dark-red. port. Haem. oedema bet. oes. and trach.		All lobes cont. air.	Hyperaem.	Haem. in walls of the alim. tract; mesent. glds. sl. enl.

TABLE II.—Continued

6.	No	Base tongue pale, Tons. enl., oedem., dark-red.	Epigl. free. haem. on vocal cords, big haem. at bifurc. trach.	Trach. glds. not markedly ch., lymph gl. on the arc. aortae embedded in haem. sw.	In lt. upper lobe two lobul. foci of hazelnut-size; in lt. lower lobe one. Rt. lung extensive hepatitis. Rt. pleuritis with punctif. necros.	Hyperaem.	Haem. in myocard.
7.	No	No marked ch.	Much haem., esp. on post. wall. Tracheitis Haem. oed. bet. oes. and trach.	—	Haem. along big bronchi, all lobes cont. air.	Hyperaem. sl. enl.	Mesent. glds. dark-red, sl. enl. Punctiform haem. in walls small intest.
8.	No	No marked ch.	Haem. in larynx and trach., esp. on post. wall. Haem. oed. bet. oes. and trach.	Embedded in haem.	All lobes cont. air.	Small, pale.	Haem. in walls of aorta and myocard. Liver chocolate col.
9.	No	Haem. on muc. memb. of nose; tons. not ch.	Haem. in larynx and along whole trach., esp. on the post. wall.	Dark-red, enl.	Partial fibrin. pleur. Confl. hepatitis. in lt. lung, except apex and lower margin. In upper part of lt. lower lobe big. hep. area.	Consid. incl., soft.	Extens. haem. at radix aortae and pulmon. veins. Haem. in myocard. and walls of the blad.
10.	Submax. lymph glds. enl. but pale; no haem.	Dark-red. sw., small haem.	Larynx. haem. In the cent. port. of trach. haem. Bet. oes. and trach. oed.	One gl. in centr. part of tr. mark. enl. On the V. cava black-red, very enl. gland.	Roots of lung dark-red; haem. around pulm. veins. All lobes cont. air.	Normal.	Haem. in the myocard. Duoden. muc. membr. detached. Intest. contents show gelat. pinkish colour.
11.	No	Fauces sl. haem. Tons. no mark. ch.	Larynx with punctif. haem.; haem. stripes along tr. On the post. wall haem. oed. Serious haem. at bifurc. trach.	Trach. glds. dark-red, enl., trach.—br. glds. surrounded by gelat. haem. oed., dark-red, enl.	All lobes cont. air; subpleural haem.	Normal.	Haem. in advent. aortae, myocard. and blad.
12.	No	No marked ch.	Spotted haem. esp. in post. wall.	Severe mediastinitis. Tr.—bronch. glds. sw., haem., dark-red.	Lt. lobes hepat. Rt. l. lobe hep. focus; rt. m. lobe small foc. Partial fibr. pleur.	Much enl., dark-red.	Haem. in myocardium.
13.	No	No marked ch.	Ac. tracheitis.	Mediast. glds. sw.	Both lungs confl. hepatitis. foci. Acute pleur.	Acute sw.	Haem. in small intest. Mesent. glds. sl. enl., dark-red. Haem. in myocard.

TABLE III

Showing macroscopic changes in inhaled sisels.

No.	Path. changes in ext. lymph-glds	Tonsils & fauces:	Trachea & trach.-bronch. glands:	Lungs & Pleura:	Spleen:	Other organs:
1.	Subm. gl. dark-red, enl.	—	Hilus-glds. enl., hyperaem.	All lobes contain air.	—	No changes.
2.	—	—	Tr. gl. dark-red, enl. Haem. oedem. in mediast.	Lungs with hyperaem. streaks. Punctif. haem. in pl., small pn. patches. Pleuritis	Acute sw.	Haem. in myocard., kidneys infl.
3.	—	—	Oedema of mediast., glds. enl.	Small pneum. patches. Pleuritis.	Acute sw.	Haem. in myocard. and the intest. serosa.
4.	—	—	Oedema of mediast. Glds. sw.	Pneum. patches size of pea.	—	Small intest. dark-red. Mes. glds. unchanged.
5.	Subm. lymph. gl. sl. enl.	—	Whole mediast. black-red. oedemat. mass.	All lobes pneum. confl. patches.	—	Dark-red masses in small intest. but no distinct haem. Mes. glds. unchanged.
6.	—	—	Oedema of mediast.; glds. sw.	All lobes contain air.	—	Like sisel 5.
7.	—	—	Colorless oedema bet. oes. and trach. Glds. sl. enl.	Some pleur. patches. Many confl. pneum. patches (50-100).	Acute sw.	No changes.
8.	—	—	Haem. in trach., mediast. glds. embed. in large haem.	Rt. lower lobe, and parts of lt. lung hepat. Pleuritis.	Acute sw.	Mesent. glds. sl. sw.
9.	—	—	—	—	—	Infl. of alim. tract.

TABLE IV. (GROUP II)
Showing Tarabagans Killed—at Varying Intervals After Inhalation.

No.	Size	Inhaled with lung emulsion from	Killed after	PATHOLOGICAL CHANGES IN					Spleen.	Other organs.	Heart blood	Tests on guinea pigs.
				Cut. lymph glands.	Tonsils	Trachea	Tracheal and tracheobr. glands.	Lungs and pleura.				
1.	Big	Tarb. 11	24h.	None	None	None	None	Some punctiform haem.	None	None	sterile	9 gps. inoc. cut. or subcut. with emuls. of different organs rem. healthy
2.	Small	Tarb. 12	30h.	None	None	Muc. memb. sl. hyperaem.	None	None	None	None	sterile	Gp. inoc. with cult. from mucous memb. of trachea rem. healthy.
3.	Med.	Tarb. 13	42h.	None	None	None	None	6-8 small pleural haem. with diam. 1-1.5mm.	None	None	sterile	not performed.
4.	Med.	Tarb. 13	45h.	None	None	None	None	None	None	None	sterile	not performed.
5.	Very big	Tarb. 13	51h.	None	None	None	Gelat. oedema between bifur. tracheae and aorta.	None	Slight hyperaemia	None	sterile	not performed.
6.	Med.	Tarb. 12	52h.	None	None	None	Trach-bronch. glands sl. enl., embedded in a gelat. oedema.	None	Slight enl. hyperaemia	None	pos. (about 100 germs in 0.1 c.c.)	1 gp. inoc. subcut. with cultures from the heart-blood died after 11 days; P.M.: suppurated inguinal buboes, secondary pl. pneum.
7.	Big	Tarb. 12	72h.	None	None	Small haem. around vocal cords. hyperaem. muc. memb. of tr.	Trach-br. glds. much swollen, embedded in a haem. oedema	Hyperaemic. One small haem. at periph. left upper lobe suffusions on pleura.	Slight enl. hyperaemia	Muc. memb. of stomach and small intest. hyperaemic. Mesent. glands changed. Kidneys much congested.	abundant growth.	1 gp. inoc. with cultures from heart blood died after 6 days, 1 gp., inoc. with bact. cult. from lungs died after 4 days. P.M.: acute bubonic plague.

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PLAGUE TRANSMISSION THROUGH THE ECTOPARASITES OF THE TARABAGAN

BY WU LIEN-TEH, J. W. H. CHUN, R. POLLITZER

In the summer of 1923 it was definitely established by our bacteriological investigations that Siberian marmots (tarabagan—*Arctomys bobac*) are subject to big epizootics of plague and hence undoubtedly responsible for the frequent human outbreaks in Transbaikalia. Up to that time the attention of the workers in those parts had been absorbed by the investigation of this main problem both in the fields and the laboratory, and little attention was paid to side issues. Now the opportunity had come to attempt a solution of problems of this category, most prominent among which seemed to be the part played by the tarabagan parasites in the spread of plague infection. An active rôle of those parasites could not be taken for granted *a priori*, at least as far as the spread of infection from animal to man was concerned, because the trapping, killing and skinning of the valuable animal give ample opportunities for direct contact; and indeed in most of the outbreaks on record such a direct infection was at least probable and in not a few even certain. On the other hand, it had to be borne in mind, that the parasites, including the fleas, stick with a remarkable tenacity to the corpses and even to the pelts of the animals. Hence it was of great practical importance to determine whether they might harbor the infection or not.

The Tarabagan flea (*Ceratophyllus silantievi*)

Apparently the first author who emphasized on theoretical considerations a possible rôle of the tarabagan flea (*Ceratophyllus silantievi* Wagner 1898) was Tiraboschi (1904). Dudchenko (1909) mentioned that the tarabagan fleas could bite man; he recorded no experiments done in this respect and it would appear that he relied solely upon the communications of the hunters; his statement that after the bite, a red spot appears at the site certainly cannot be generalized. One of us (W. L. T. 1913) proved that the fleas readily bite man under experimental conditions. Dudchenko saw in 1915 one of his burrow-diggers bitten on the neck by a tarabagan flea. This same observer pointed out that the tarabagan fleas—like those of the other Transbaikalian rodents—are able to emancipate themselves from

their hosts for considerable periods, during which time they vegetate in the fields. Jettmar (1922) described a human plague outbreak, apparently caused by such "free living" fleas, because the group of hay harvesters decimated by the pest had in all probability not been in contact with tarabagans, but had been much molested by fleas. The suspicion thrown upon the *Ceratophyllus silantievi* by this and a few other outbreaks with a more doubtful anamnesis was much strengthened by Sukneff (1922) who saw bipolar staining plague-like bacilli in a flea, picked up from a naturally-infected tarabagan. This, however, could not be confirmed by cultural or experimental tests. Furthermore, a second series of biting experiments (1923) by us convinced us that the fleas do not defecate when biting, and hence nothing short of an actual transmission of the disease by means of those parasites could prove their rôle.

We started such biting experiments in the spring of 1923 in Harbin by letting one supposedly infected tarabagan flea feed upon a healthy tarabagan and a healthy guinea pig. Both animals survived. The flea, which had bitten an infected animal on May 31st died on June 6th without showing any evidence of plague infection *post mortem*. It was evident that no successful flea experiments could be carried out in Harbin, because the tarabagans, already almost flea-free when they reach here after a long journey by rail, soon lose the few remaining fleas in the cages. Therefore when we had the opportunity to work with our Russian colleagues in Transbaikalia, we suggested to them a continuation of such experiments on the spot. They consented readily and the following two experiments were performed jointly by Chinese and Russian workers.

(a) On a tarabagan, artificially infected 3 days previously by injection, 32 *Ceratophylli silantievi* were exposed on August 23. The animal succumbed to plague early on the 27th. Before it was removed from its cage a healthy tarabagan was exposed in the second compartment of the cage, separated by a wire screen. After 3 hours a guinea pig was placed for half an hour on the dead tarabagan. Then the dead animal was searched for fleas and 7 live ones were recovered which were placed on the above mentioned healthy tarabagan. Neither this tarabagan nor the guinea pig became infected.

(b) From a naturally infected tarabagan, found on August 24, 22 fleas were recovered besides 13 lice. 13 of the fleas were placed on a healthy guinea pig; a second guinea pig was injected with an emulsion of 6 fleas, a third one with an emulsion from

the feces of three fleas obtained on the 27th and 29th of August. Those three animals, as well as a fourth guinea pig which had been placed on the tarabagan before all the fleas had been removed, survived. This result was the more disheartening as the lice collected on the naturally infected tarabagan contained virulent *B. pestis*.

A third experiment was performed by Sukneff, after our departure, by exposing on an artificially infected tarabagan 200 tarabagan fleas. When the animal died, a healthy tarabagan was exposed in the adjacent compartment for 3½ hours. Then the dead body was searched for fleas; only 8 of the 200 could be found, 7 of which were exposed on the above mentioned healthy tarabagan. This animal survived. Sukneff saw, however, many plague like bacilli in the crushed 8th flea, and a guinea pig injected with part of its body died of plague.

(c) *Successful experiment.* Thus there could be no more doubt that the tarabagan fleas can actually contain living and virulent plague bacilli, and the rather discouraging results of the 1923 campaign could not shake our firm belief in the infective rôle of the fleas and our hope to overcome gradually the great difficulties of those experiments. With this object in view, we started by ourselves a new series of experiments in August 1924, choosing as our base on this occasion our well equipped laboratory at Manchouli Hospital, where Resident Medical Officer, Dr. Li An, assisted us.

The first two experiments (placing three fleas and one flea respectively, collected from animals succumbing to artificial infection, upon a tarabagan) gave no positive result; the tarabagan (No. 309) died 6 days after the second exposure, but showed at autopsy no plague, its death being due to a streptococcal infection from a wound on its right hind leg caused by the snare. It could be clearly seen that it was very difficult to keep these wild tarabagans alive under the strict confinement necessitated by the experiments, and we resolved to perform the next test with guinea pigs only. We think that the positive result of the third experiment is due to this decision. The following are the details:

(d) A healthy guinea pig (No. 311) was infected cutaneously with one loop of a virulent culture on August 22. Soon after infection 17 tarabagan fleas were fed upon it. The guinea pig was found dead on 27th in the morning. Before it was handled, a healthy guinea pig, kept up to that time in a room far away from the laboratory, was placed in a clean bucket. Then guinea pig 311 was searched for fleas; five lively

tarabagan fleas, found upon it, were collected in a clean test tube and exposed on the healthy guinea pig, the greatest care being taken not to touch the animal or the inside of the bucket. Only then was the autopsy on guinea pig 311 performed; this revealed typical plague (right inguinal bubo) with marked septicemia. The guinea pig upon which the five fleas had been exposed (No. 313) was afterwards kept in the laboratory; an accidental infection of the animal is out of the question, because no more plague experiments were performed and there was no previously infected animal in the room. The animal began to sicken on September 2 and showed on the 4th symptoms of plague infection. Being unusually strong, this male animal managed to survive up to the morning of the 6th. The autopsy showed a small right inguinal bubo and otherwise typical signs of subacute plague (large spleen with many nodes, the largest being bigger than the pea-sized nodes in the lungs). Smears and cultures from the organs were positive, numerous plague bacilli being seen in the bubo and spleen. The whole guinea pig is now preserved in our museum. Another guinea pig, 316, infected cutaneously with the spleen culture on September 8th, succumbed after 4 days, showing at autopsy right axillary bubo and other signs of plague.

We can assert that this one experiment forms positive proof that *Ceratophyllus silantievi* is capable of spreading plague infection from one animal to another. It would be highly desirable to supplement this knowledge by observations upon the flea rate and upon the density of the flea infestation in the different tarabagan districts. It can be inferred from casual observations that the flea rate, which is seemingly not very low immediately after hibernation, decreases in spring and early summer, and increases in the latter part of the summer. Some authors state that the tarabagans free themselves to a large degree from their fleas before they begin to hibernate. The only exact observations were made by us in the spring of 1923 and in the summer (August) of 1924 upon tarabagans transported after their capture for about 40 miles to Manchouli. Hence our figures are certainly too low, especially those of 1924, when the animals were caught under difficult conditions. We found the 1924 summer flea rate only slightly higher (1.5 against 1.3) than that in spring 1923. Further investigations upon this whole problem, including the question of the "free living" fleas, which has never been properly undertaken, seem very necessary. The practical difficulties accompanying such observations on a large scale are, however, well nigh insurmountable in those bare regions. It will be easier to study infected fleas both *in vitro* and *post mortem*. Thus far we could spare no living fleas for such tests, and the few dead

specimens found on the infected animals were not well enough preserved. Neither living nor dead fleas were found on guinea pig 313.

The Tarabagan Louse*

Experienced hunters assert that the tarabagan lice can crawl upon human beings from freshly captured animals, and also that they remain alive on the skins for weeks (Jettmar, 1922). Wassilewski (1921) actually saw a louse crawl from a tarabagan corpse on his knee. The first exact observation was made by us in the spring of 1923, when we succeeded in causing a louse, starved for three days, to bite one of us. In 1922 Sukneff saw plague-like bacilli in smears from 3 lice taken from a natural plague-infected tarabagan. In the summer of 1923 we and Sukneff working in Siberia found 13 lice on a tarabagan which had succumbed to natural plague infection; a film made from one of these showed a considerable number of plague-like bacilli. A guinea pig injected with an emulsion of three of the lice succumbed after six days to typical plague (bilateral inguinal buboes); a second guinea pig upon which 10 living lice were placed, survived. The positive results obtained with these lice are the more remarkable, as the fleas of the same tarabagan were to all appearances free from plague infection. After our departure Sukneff made similar experiments with lice from an artificially infected tarabagan and obtained again positive results in smears and by injection. Controls of 8 lice upon a healthy tarabagan were negative. Jettmar (1924) made an exhaustive study of plague infection of the tarabagan lice. He found that the lice suck the blood of man, as well as that of the ground squirrel and guinea pig, but will not live on a strange host for any length of time. When the lice suck plague blood, the bacilli grow rapidly in clusters, and finally fill up the lumen of the stomach; the lice die after two or three days of the infection, displaying a characteristic red color, due to a sedimentation of hemoglobin; the feces of the lice contained large numbers of plague bacilli in pure culture, while none were found outside the intestinal tract; the lice were found to contain virulent plague bacilli for at least 13 days. Jettmar finally succeeded to infect a healthy sisel (?) by placing upon it 40 tarabagan lice collected 24 hours previously from a dead artificially-infected tarabagan.

*We do not give a Latin name in the text, because there still exists a controversy about the species of the tarabagan louse. The Institute consulted by us determined this louse as being *Haematopinus lyriocephalus* Burmeister, whereas another authority classed it as a *Linognathoides spermophili* Cunnings 1914. A third determination is not yet concluded.

The tarabagan tick. (*Rhipicephalus* (*R. haemaphysaloides*?].)

So far we have not been able to induce this tick to bite man, even after a starvation of almost three weeks. The ticks seem to disappear quickly when placed upon a guinea pig. A few experiments done with plague infection gave negative results; neither a tarabagan, upon which live ticks, collected from a plague injected tarabagan, were placed, nor a guinea pig injected with an emulsion from such ticks succumbed to plague. Though perhaps—even when a rôle of the ticks can be taken for granted—they may not spread plague infection *actively*, the insects which stick tenaciously to corpses and skins, might serve as *passive* spreaders of infection. For this reason these experiments will be continued.

Conclusion

Now that we have finally succeeded in infecting a healthy animal by means of tarabagan fleas, it may be stated that they undoubtedly play an important part in the spread of the infection, not only from animal to animal, but also from animal to man; we are convinced that they were involved not only in the constant Siberian outbreaks, but also in others where a direct contact with infected animals occurred. The lice are certainly an important link in the propagation of the disease from animal to animal—our observation on the lice and fleas from one and the same naturally infected tarabagan is certainly illustrative in this respect. It is at present difficult to gauge how far the lice actually spread the disease to man. A few outbreaks, where the affected had contact with tarabagan skins only, seem somewhat suggestive of their rôle. Our observations, made both on the fleas and the lice, thus show that any attempt to control the handling of tarabagans and the trade with their skins must include not only bactericidal, but also insecticidal measures.

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SOME REMARKS UPON THE EPIDEMIOLOGY AND HISTOLOGY OF PNEUMONIC PLAGUE

BY H. M. JETTMAR

(Summary of an article appearing in the "Archiv fuer Schiffs- und Tropenhygiene," 1925, 29, 650)

Owing to the strict measures which were taken to prevent the importation of the pneumonic plague epidemic raging during the winter of 1920-21 in North Manchuria into the adjacent Transbaikalian territory, only a few limited outbreaks and isolated cases were noted in this Russian province. Some of these proving of considerable interest are herewith discussed:

1) The outbreak at Kailastui.

On January 25, 1921, a Chinese suffering from pneumonic plague arrived from the heavily infected Dalainor coal mines in the Manchurian settlement situated on the Argun River opposite Kailastui. His house was visited by a Cossack who then returned to the Russian village of Kailastui. Here he fell ill on January 30 and died on February 2. All the ten persons in close contact with him (they all shared the common living room of a hut) succumbed to pneumonic plague.

The sister of the first victim who had only come to see his dead body, had to be isolated in this hut together with the remaining contacts. Supplied with proper instructions and a mask she remained well.

Another case in Kailastui occurred in the person of the sister-in-law of the above Cossack who had visited him on February 1. She fell ill during the night of February 3 and died about 48 hours later. The hut where she lived had also only one living room, yet her four contacts remained well though taking no special precautions. However, contrary to the Cossack she did not cough into the room but—lying in a bed separated by a low wooden partition from the rest of the room, she coughed towards the wall.

2) Observations upon a pneumonic plague victim committing suicide early in the disease.

A peasant living in Klitshki village who had been perfectly healthy up to February 18, 1921, was seized by rigor on the

evening of that day. He soon complained of headache as well as stabbing pain in the chest and spent the night near the stove. Towards dawn he left the hut and hung himself on the fence round the compound where he was found six hours afterwards by the plague prevention staff.

Since his body was frozen stiff it was not possible to perform a complete *post mortem* examination. The lungs were, however, inspected *in situ* and pieces of this organ as well as the spleen were preserved for histological examination.

Inspection of the lungs showed hepatisation of the right middle lobe as well as confluent pneumonic foci in the right upper lobe. Early involvement appeared to be present in the lower lobe while the left lung seemed free.

The results of histological examination of

- I) Pieces from the right middle lobe showing hepatisation;
- II) A big bronchus with adjacent parts of the lung;
- III) The left lung;
- IV) The spleen

may thus be summarised:

- 1) The alveoli are quickly filled with a bloody-serous and specially a cellular exudate so that already on the first day of illness whole parts of the lungs are hepatized. The bacteria are at first evenly distributed in this exudate but soon show a marked affinity for the alveolar walls adjacent to small veins.
- 2) Phagocytosis of the plague bacilli may be fairly frequently noted but is restricted to the epithelial cells containing inhaled dust (Staubzellen).
- 3) The cells of the exudate consist at first of almost exclusively erythrocytes, then a greater number of epithelial cells (Staubzellen) appears and finally leucocytes. When hepatisation has become complete, the latter form the bulk of the exudate. A part of the initially exudated cells shows disintegration of their margins followed by granular fragmentation, plague bacilli immigrating into the separated particles.
- 4) Fibrin is plentiful in a few alveoli only.
- 5) The capillaries and blood vessels are completely sterile and much congested. Mast cells are often present. Isolated haemorrhages caused through rupture of the capillary wall and abundant diapedesis of erythrocytes are noted.

- 6) The walls of the blood vessels in the affected areas show in places where the plague bacilli in the lymph spaces have come quite near to the endothelium, proliferation, swelling and desquamation of the endothelium. Penetration of the bacilli into the lumen is however nowhere seen.
- 7) Thrombosis in capillaries is not seen.
- 8) The bronchi show degeneration and desquamation of the epithelium as well as hyalinization of the basal membrane, infiltration with leucocytes and oedema of the walls, mucilaginous degeneration of the mucous glands and sometimes disintegration of the elastica.
- 9) Mast cells are numerous in the lung septa and the peribronchial tissue.
- 10) Perichondrium and cartilage of the bronchi show no morbid changes.
- 11) The spleen shows acute infectious hyperplasia. It contains no bacteria.
- 12) Hyalin is found in the alveolar walls, further in the alveoli situated near the small vessels where it forms strata and trabeculae near places rich in bacteria.

It is also found in the basal membrane of the bronchi and in the walls of a few pulmonary and spleen vessels.
- 13) Contrary to the later stages of the disease necrosis is absent.
- 14) The plague bacilli seen in the tissues show characteristic form and polar staining. The bacilli lying in disintegrated tissues show mostly globular form but normal size. Marked involution form is displayed solely by the bacilli phagozyted by the epithelial cells.
- 15) Bacteria are nowhere noted within the lumen of the vessels. They are numerous in the lymph spaces of their walls as well as those of the peribronchial tissue.
- 16) Besides the *B. pestis* no other bacteria are noted in the tissues. (This is in contrast to the majority of other cases observed in the same outbreak which were dissected in a more advanced stage of the disease).

The scanty material recovered from this case does not permit of final conclusions as to the spread of infection in the body. However, the severe changes in the alveoli and bronchi in this early stage of illness speak for a primary localisation in the air passages of the lungs.

On the other hand no findings are made indicating a haematogeneous spread of infection (as claimed by Kulescha & others). The blood vessels, especially the capillaries of the lungs are sterile and alterations of the vessels, if present, are comparatively slight.

NEW PNEUMONIC PLAGUE WARD, HARBIN 1926

BY Y. M. KWAN

Among the resolutions passed by the International Plague Conference of Mukden 1911 was the following:

“The need for isolation of pneumonic plague patients being urgent, permanent isolation hospitals should be available. Such isolation hospitals should admit of individual isolation, be of rat-proof construction, and be capable of easy disinfection. In the grounds of such isolation hospitals ample space should be provided for the construction of additional emergency wards, for which purpose the site should be prepared and foundations laid. The provision of ample air space and light is desirable.”

It will be seen that very few details were given as to method of construction of a pneumonic plague ward. In the summer of 1912, when building operations for the new plague prevention hospitals were hurriedly started, three of the five plague wards were so designed as to admit of individual isolation, but owing to the lack of sufficient cement at the time the floors were all made of wood. In each unit is a long corridor, four feet wide, from which a series of twelve doors open into their respective cubicles, each with its own outside window. A small glass window, one foot square, is inserted in each door to enable the nurse or attendant to watch the patient from the corridor. The physician or nurse attending the patient must enter the chamber and thus be exposed to great risks and discomfort.

In our new pneumonic plague ward constructed in 1926 we have obviated the above weak points and added several improvements for investigation and research, so that this new building may be considered as near a model as any such can be. A description now follows:

The new building is rectangular in shape, 78½ feet long, while the breadth has a maximum of 55 feet and a minimum of 46 feet. Foundations are 7 feet deep. Two entrances lead into the building. The first, patients' entrance, (in the center of its longest part) opens into a square corridor (10 × 10 feet), from the left of which a passage leads to the attendant's

The author, a valued member of the staff, succumbed to phthisis at Canton in February, 1934.—(Ed.)

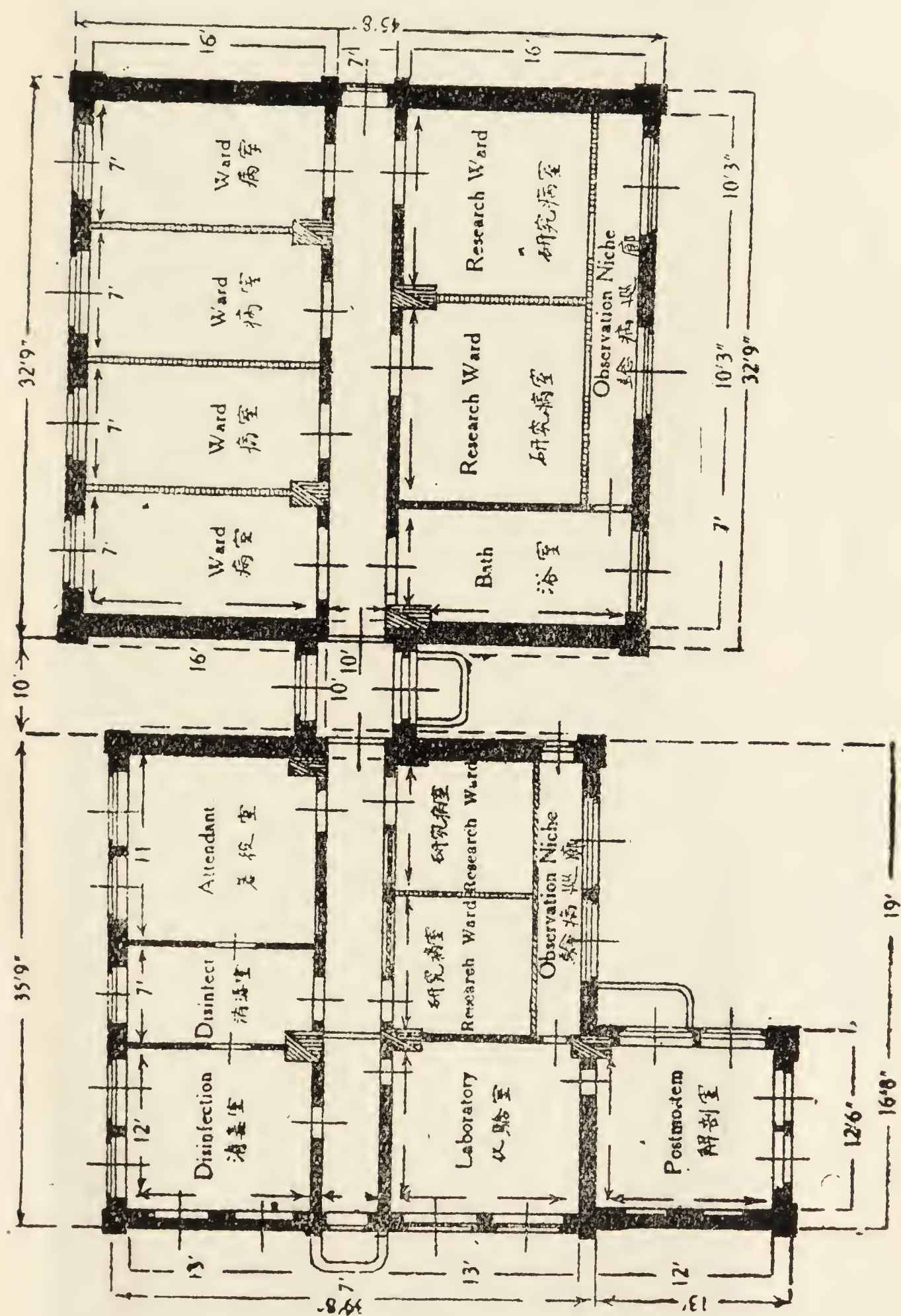
room and two disinfection rooms on one side and two research wards and the laboratory on the other. These rooms can also be reached from the second entrance at one end of the long corridor by the medical staff. From the laboratory a door leads to the postmortem room and thence into the separate exit. Between the laboratory and postmortem room is a sliding glass window, similar to that often seen in an operating theater for communication between workers on the two sides. The floors are all constructed of cement slabs on a crushed brick base. The windows are all very large:—4 measuring 7 ft. by 4 ft. 8 in.; 3 meas. 7 ft. by 5 ft. 8 in.; and 13 meas. 7 ft. by 4 ft. Total 20 windows.

Each of the four big cubicles for a plague patient is 7 feet wide by 16 feet long and 11 feet high. The most interesting features of this new Pneumonic Plague Hospital are:

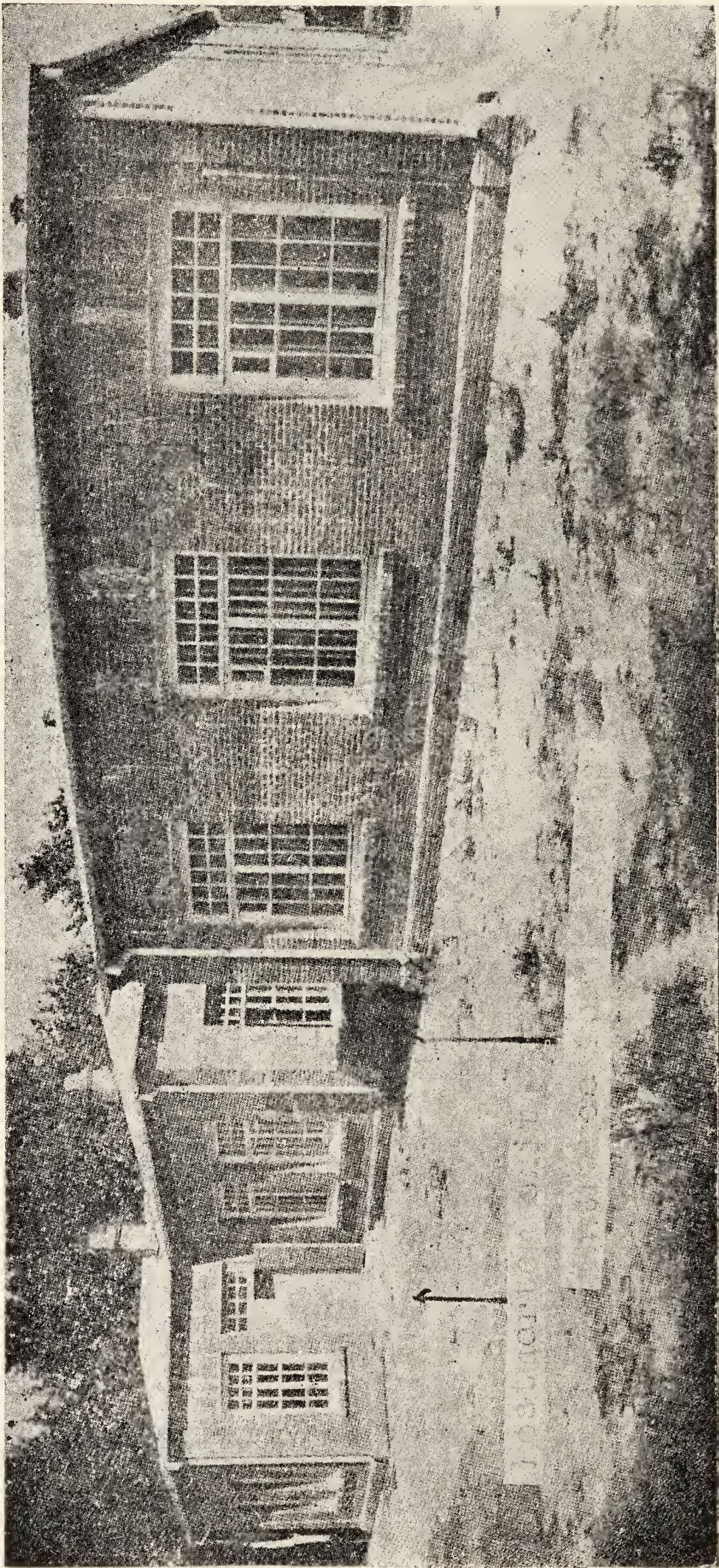
- (a) The adjoining *Laboratory and Postmortem Room*. Where the course of disease is so rapid, every minute saved is important, and the presence of the laboratory side by side with the wards will enable the medical staff to prepare all materials for diagnosis and treatment on the spot. When a patient dies, the same facilities for investigation are also at hand.
- (b) The *observation niche* measuring five feet wide and separated from the individual cubicles by a wide glass partition, which reaches as far as the ceiling. The advantages of this niche are:
 - i. The physician can observe and conduct investigation upon the patient at all times without being in the same room and breathing the same air.
 - ii. By means of a small sliding glass pane in the partition three feet above the floor, the patient's arm can be extended into the niche for blood examination, serum injection etc. without risk to the physician. There may even be no need for him to wear a mask since complete security is assured without hindering any view.
 - iii. The partitions dividing the cubicles can be removed at a moment's notice so as to form a large room for saline injection in the treatment of cholera cases.

The addition of these two niches in the new Plague Ward, one through the laboratory and the other through the bath room, will no doubt further facilitate any investigation upon pneumonic plague patients that we may desire when the next epidemic breaks out.

模範的肺疫病室(哈爾濱東三省防疫總處院內
一九二六年落成)之平面圖



NEW PNEUMONIC PLAGUE WARD, HARBIN HOSPITAL 1926.
濱江防疫醫院模範肺疫醫病室



ENDOPARASITES OF THE TARABAGAN

By LI YUAN-PO

Examination of tarabagans captured in Mongolia for the purposes of the Manchurian Plague Prevention Service led to the discovery of the following intestinal parasites:—

1. *Ascaris* sp. nova.

The body of this worm is grayish-white in colour. The head is provided with three lips, one upper and two lower; the papillae are relatively large. The cephalic extremity is quite different from that of *Toxastrus limbatus* as found in dogs and cats, the head in the latter case being provided with membranous wings.

On the other hand, the ascaris of the tarabagan has a shape resembling in general that found in the human intestine. But the caudal extremity of the female is papillar—a feature different from any hitherto described.

- A. *Male ascaris*, 8 cm. long, 2 mm. at widest part. Thinner and shorter than the female. It was decomposed when found, so that the anatomy is not quite clear.
- B. *Female ascaris*, 10 to 17 cm. long, 2.5 to 3 mm. at widest part (two specimens). The vulva is located at the anterior fourth of the body. Its prominence can be seen with the naked eye. Microscopically, it looks like a mortar, concave in the centre, thus forming a round cavity. The ovaries of the human ascaris look like a spiral tube winding round the body. In the new ascaris this is missing. The papillar shape of the tail is also peculiar, since the tip, where the rectum opens, is surrounded by spiral muscular tissues for elongation and contraction. The connection between the vagina and uterus can be clearly seen under low power. Otherwise the features are much the same as in the human ascaris.
- C. The *eggs* are gray in colour and not different to those of the human parasite. In the faeces of the tarabagan we found ripe ova, 60-66 μ long and 48-54 μ wide. The measurements of ten ova examined are as follows:—

Breadth: 54 50 54 48 54 48 52 60 54 54 Av. 52.8 μ .
Length 66 60 66 60 63 66 60 66 72 66 Av. 64.5 μ .

This ascaris has also been seen by Sukneff in Chita.

2. *Entamoeba bobaci* sp. nova.

We found in the faeces of 7 of our 43 tarabagans besides *Trichomonas*, *Trichomastix*, *Hexamitus* and *Chilomastix* a peculiar species of amoebae. This is generally found in the coecum and colon and closely resembles *Entamoeba coli*.

- a. *Examination of fresh specimens*.—In fresh preparations its movements are comparatively slower than those of *Entamoeba dysenteriae* and its pseudopodia more hyaline and bulkier than those of *Entamoeba coli*; in non-motile forms no distinction can be made between ecto—and endo-plasma. The nucleus is almost always visible during locomotion.
- b. *Examination after staining*. (Iron haematoxylin after fixation in Bouin's fluid).—The dimensions of the Amoeba are usually 20-35 μ ; one finds specimens of 15-40 μ , i.e. corresponding to the size of *Entamoeba coli* (20-30 μ after Brumpt & Dobell). Measurements of 100 amoebae showed a mean of 24 μ .

The granular protoplasm is comparatively paler and less dense than that of *Entamoeba coli* and *E. dysenteriae*. A great number of vacuoles is met with containing various substances ingested by the amoeba. Sometimes the pseudopodia are very big as in the case of *Entamoeba muris*. The tarabagan amoeba is very voracious: one finds in its vacuoles all sorts of objects, like bacteria, yeast cells, *Hexamitus*, *Eutrichomastix*, the latter dead or degenerated—in one word all the normal contents of the tarabagan intestines but never red blood corpuscles. About 60% of the amoebae contain as parasites a species of *Sphaerita*. Such parasitism was first described by Dangeard and subsequently found by different authors (Leger & Duboscq, Chatton & Brodsky, Dobell, Wenyon, Noeller, Brug, Mattes & Brumpt, etc.) in various amoebae. However, rarely such a high percentage of parasites has been observed. These parasites, spherical in form, differ very widely in size, from being just visible up to 2.5 μ ; their number varies from 1-40. The destruction of the amoebae through the *Sphaerita* has been demonstrated by various authors. We have observed that at a certain stage of development they cause a considerable hypertrophy of the protoplasm of the amoeba, resulting in the bursting of the latter.

Another species of *Sphaerita* with annular spores, measuring 4 μ by 2.5 μ and found by Noeller & Brug in *Entamoeba coli*, is also at times found in the tarabagan amoebae. This *Sphaerita* shows a darker color at its anterior pole, otherwise it is light brown. Both *Sphaeritae* may co-exist in one and the same amoeba.

The nucleus which measures 3-5 μ , is in general excentric or subcentric and characteristic for the genus *Entamoeba*; it is

spherical and vesicular with a peripheral chromatine layer formed by spherical granules situated close to one another.

The voluminous karyosoma is in general excentric or subcentric, rarely centrally situated. There are fine chromatic granules in the intermediary zone between the membrane and the karyosoma.

In one case we found an amoeba with a nucleus resembling that of *Karyamoebina* (Kofoid). It is impossible to decide whether this is a special form of *E. bobaci* or whether we have to do with another species.

c. *Encystment*.—In our seven tarabagans we found neither in the faeces nor the caecum or colon at autopsy any cysts, although numerous vegetative or preencysted forms were present. Perhaps encystment of this amoeba is very rare as has been observed in the case of *Entamoeba cobayae* by Holmes.

d. *Pathogenic role*.—The tarabagan entamoeba does not phagocytize red blood corpuscles and is therefore probably not pathogenic for its host. Further the caecum and colon of some animals showed no peculiar histological alterations nor penetration of the amoebae into the tissues.

e. *Classification*.—Though resembling at first glance *E. coli*, our amoeba is distinguished from it by its more hyaline and bulkier pseudopodia and the less dense protoplasm. It forms therefore a species of its own to which we gave the name of *Entamoeba bobaci* after its host.

3. *Enteromonas* sp.

We saw in the faeces of the majority of tarabagans with dysenteric symptoms a flagellate resembling the *Enteromonas hominis* da Fonseca. This *Enteromonas* is of spherical or oval form and shows extremely rapid movement, known as the 'rapid dancing movement' of Wenyon & O'Connor. It changes its form repeatedly and abruptly. It possesses neither a cystosoma nor an undulant membrane. Young flagellates show in their posterior part a pointed prolongation, 6-8 μ long. The precystic form measures 16-18 μ in length and contains numerous chromophile granula in its protoplasm. This distinguishes it from other *Enteromonas*.

The nodule is situated in the anterior part of the body. The part between the karyosoma and the nuclear membrane does not stain. Four flagellae derive from the blepharoblast situated in only one or two anterior flagellae instead of three and the posterior one passing along the body with which it is in close contact. The flagellae stain with difficulty. Sometimes one sees only one or two anterior flagellae instead of three and the posterior

one stains only in its intra-protoplasmatic portion. The precystic form encloses glycogen either in one big mass or in three or four small agglomerations demonstrable by the double Lugol method or that of Best.

Culture.

a.—The *Enteromonas* contained in the tarabagan stools can be preserved for more than a month at a temperature of 20°C. in artificial normal serum. We could not observe any division.

b.—Musgrave-Clegg Medium.

Using this medium at a temperature of 37°C. one can observe on the next morning numerous division forms.

Degeneration is retarded when one keeps the cultures at a temperature of about 25°C. In this case division of the flagellates commences after 3-4 days.

c.—Ovo-mucoid medium of Hogue.

d.—Tarabagan serum (diluted 1:10 with 1.5% salt solution).

The flagellates rapidly multiply in these two media but their degeneration is also precocious.

Pathogenic Role.

These flagellates are constantly found in varying numbers in the stools of all tarabagans with dysenteric symptoms. However, it is impossible to say at present whether they are the actual cause of this condition or merely "*germes de sortie*." Further investigations are called for.

Conclusion.

In recapitulation one can find in tarabagan stools

- 1.—*Ascaris* sp.;
- 2.—*Entamoeba* sp.;
- 3.—*Enteromonas* sp.

Furthermore one notes sometimes the presence of cysts resembling those of *Blastocystes hominis*.

THE PERPETUATION OF PLAGUE AMONG WILD RODENTS*

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CONTENTS

I.	Introduction
II.	The ground squirrel of California
III.	The South African wild rodents
IV.	The South Russian rodents
V.	The Alpine marmot
VI.	The tarabagan
	Introductory remarks
	Earlier hibernation experiments
	Plan for new hibernation experiments
	Experiments in the winter of 1926-1927
VII.	Summary and conclusions
VIII.	Bibliography

I. Introduction

As in other epidemic diseases, one of the fundamental and fascinating problems in plague is the question of the mechanism by which the malady is carried along from season to season, often without the presence of human cases and manifest rodent disease. In this report I intend to deal with wild rodents, but for the sake of comparison it may be worth while to consider briefly the conditions among domestic rats.

In their summary on the etiology and epidemiology of plague (1) the Indian Plague Commissioners came to the conclusion that the periods between the epidemics are bridged over by cases of *acute* rat plague, the epizootic being kept in check by, (a) a high mean temperature, (b) by a diminution in the total number of rats, together with an increase in the

*In the preparation of this article I have received much assistance from Dr. Robert Pollitzer of our staff. To him I wish to accord my best thanks.

proportion of immune to susceptible animals and (c) by a diminution in the number of rat fleas.

In an earlier contribution (2) the Commissioners had already presented evidence to refute the theory that cases of chronic (or resolving) rat plague are important in the propagation of the infection. Among the reasons given are that it is unlikely that rats contract acute plague by feeding upon the carcasses of chronic plague animals, and that there is no evidence that resolving plague lesions "light up," resulting in bacteremia and rendering the rats dangerous. The Commissioners therefore conclude "that these chronic plague rats, inasmuch as the bacilli are shut up in abscesses where fleas cannot possibly get at them, are *per se* of no importance in spreading the infection" (1).

Since this statement was made in 1908, we find frequent references to the probable rôle played by chronic plague rats. To our knowledge no satisfactory evidence has been brought to support any such claims, and it may still be maintained that resolving rat plague is not an active factor in the propagation of the disease. But it is probable that sporadic cases of acute rat plague are not the only ones which keep the virus alive during the off-seasons. Recent investigations especially by Williams (3) suggest that "carriers" of plague may exist among rats, which, apparently surviving for a prolonged period, yet are suffering from bacteremia and may therefore spread infection through their fleas. A further possibility is that rat fleas which under suitable conditions are apt to remain alive, and infective for a considerable time may help to preserve the virus.

Turning now to the wild rodents we find that more or less satisfactory evidence is available for a few species only. Among those hitherto investigated, their blood sucking ectoparasites, especially fleas, have been found to be the principal, if not the only means of transmitting plague. Therefore animals with bacteremia are an essential link in the propagation of this infection.

II. The Ground Squirrel of California (*Citellus beechyi*).

Two factors favoring the spread of plague among ground squirrels are (a) the absence of a seasonal prevalence of the disease, heavy infections being noted in winter as well as in summer (4), and (b) the restriction of aestivation or hibernation principally to adult animals. Though this tendency to aestivation or hibernation (combination of both?) is noted in ground squirrels, adult animals are chiefly affected (5). The young animals suspected by Harrison (6) to be most susceptible to acute plague, keep awake throughout the year.

An important means of restraining the spread of plague among squirrels is the immunity usually developing in them in regions where the disease has prevailed for several years. McCoy (7) in an early contribution to this subject says that this "may mean a gradual extinction of the disease, or it may indicate that this partially resistant race of rodents will, if not vigorously attacked, perpetuate the disease for many years." There is no doubt that the infection tends to persist rather than to disappear. The immunity prevailing in endemic localities is not absolute as has been shown by the instructive experiments of McCoy (4) upon squirrels from plague-affected and plague-free zones respectively:

TABLE I

	Total	Died		Killed	
		Acute plague.	Subacute plague.	Residual bubo.	No lesions.
Squirrels from plague-affected zone	16	1	3	3	9
Squirrels from plague-free zone	19	8	9	—	2

It can thus be seen that subacute and even acute plague, though rare, is not entirely absent among squirrels from plague-affected zones. A similar variation in the lesions is also seen in naturally infected squirrels. Those with acute disease and some of the animals with subacute disease show bacteremia, and are therefore infective. Moreover, it is claimed by Rucker (8) that even in regions where the disease has existed for a few years and where the majority of animals present non-acute plague, small areas will be found, e.g. in isolated valleys, where the acute form predominates. For all these reasons it is not difficult to understand why plague persists among the California wild rodents.

III. The South African wild Rodents

A very rapid type of infection is noted among the wild rodents involved in South African epizootics. As far as our present knowledge goes, chronic plague does not exist among gerbilles or multimammate mice. There is little information in regard to carriers as only one Namaqua gerbille and one Karroo rat were found in the same locality which could be regarded as such. It is evident therefore that, once plague infection is introduced into a locality, many animals succumb quickly and little fuel remains to keep the virus alive.

A satisfactory explanation for this situation has been given by Pirie (9) who proved that plague infection could be maintained in a scanty rodent population by fleas. In a series of well planned experiments he demonstrated that fleas could 'carry over' the infection for a period of three or perhaps four months. He reasons thus: "The mechanism of the persistence of plague in the wild rodent reservoir is therefore not difficult of explanation, even when the rodent population is scanty. When the rodents are few in number the disease would of necessity be of a quiet, smouldering type, through frequent failure of the necessary contacts to be established. A rodent infection, however, at two monthly intervals, would be sufficient to keep the disease alive. As the number of rodents increased and the fleas presumably increase *pari passu*, so would contacts be more easily made and the number of cases increase. Whether the increase in numbers to a high level is in itself sufficient to bring about the recurrence of definite epizootics every 3-4 years, and still more markedly every 10-11 years, in accordance with Elton's theory, or whether some extra factor, possibly a widespread climatic one, must also come into play to allow of a flare-up from the smouldering stage to that of the raging fire, must be left for future observations to settle."

IV. The South Russian Rodents

As in South Africa, so in South Russia, several species are known to suffer from plague. Only a few are of fundamental epidemiological importance—, small susliks (sisels), domestic and wild mice. An interesting point is that no regular transfer of the infection occurs from the susliks to the mice, or *vice versa*, so that the epizootics run an independent course in each group (10).

As far as we know the problem of the perpetuation of plague among the *mice* has not been studied in detail. Chronic infections seem to occur (11) so it may be assumed that some immunity does develop in the course of the epizootics. Among the domestic mice "carriers" have been observed, *Bacillus pestis* growing in abundance though no macroscopic signs are noticeable (12). No doubt plague is constantly transmitted from mouse to mouse, and is not caused by periodic infections from the gnawing of human plague corpses (13). It seems safe to assume that the epizootics among these non-hibernating animals occur in the same way as among domestic rats.

The *susliks* belong to another group, since prolonged hibernation is an obligatory feature among them. As in the case of mice it has been assumed in the past that plague is not constantly present in susliks, infection being contracted after the hibernation period from plague corpses. But the systematic

work of Nikanoroff and his school has lately shown that such an unusual assumption is unnecessary.

Nikanoroff (10) has suggested an influence of the seasons upon experimental suslik plague. Tests were made to prove this by infecting fortnightly from the middle of June groups of thirty susliks with cultures of the same origin and virulence. Subcutaneous injection was employed in the four series inoculated. In the first series most animals succumbed quickly to acute plague, while in the other three the disease displayed less virulence. Thirty days after infection there still lived in the first group none, in the second 5, in the third 5, and in the fourth group 18.

These results have been supplemented by observations of Gaiski (14) carried out during a whole year. He infected subcutaneously 242 susliks: in 27 groups, using for each successive series a strain isolated from the preceding one. Seasonal differences were noted in two instances: (a) the mean length of illness varied, reaching its minimum (3 days) in June and its maximum (25 days) in winter, and (b) while in June and July 100 per cent. of the animals displayed bacteremia, only 60 per cent. were so found in winter and 40 per cent. in March. The other animals suffered either from pure local plague with bacilli confined to the site of infection, or from a transitory form (bacilli in the organs but not in the blood). The incidence of local plague was highest in winter (30 per cent.).

Of special interest are Gaiski's results with hibernating animals. Of 30 such susliks 21 awoke and succumbed after 2-22 days (average 8 days); 3 were killed 15 and 35 days respectively after infection and 6 succumbed after 45 to 138 days. Interesting details of these experiments are (a) that of the three animals killed, two, (15 and 35 days respectively, after infection), were well nourished and showed plague bacilli at the site of infection but not in the blood or the organs while, in the third (killed after 15 days) bacteremia was present, and (b) that of the three animals dying after 96, 120 and 138 days respectively, at the physiological end of hibernation, at least one had passed through a stage of bacteremia. At *postmortem* plague bacilli were present both in the internal organs and at the site of infection. In the other two animals these were found in the local abscesses only.

Later researches by Golov and Joff (15) indicate that the suslik fleas have probably a considerable share in keeping the virus alive; they prove that the feces of infected fleas may harbour plague bacilli for a considerable length of time (observed up to 79 days at a temperature of 7-10° C. and 92 per cent. humidity), though they were allowed to bite an infected animal once only. Infected fleas which were after-

wards kept starving at temperatures corresponding to those of the burrows in winter, survived up to 206 days and yielded at death living and virulent plague bacilli. Golov and Joff also showed that suslik fleas kept in test tubes at such temperatures remain active and able to feed upon hibernating susliks. Similar observations were also made under natural conditions. Suslik fleas can stand low temperatures (down to -25° C.) and prolonged starvation in empty burrows (up to 10 months).

It is thus seen that conditions for preserving the plague virus among the rodent population are almost ideal. As pointed out by Gaiski, however, the seasonal changes of susceptibility may protect the species from extinction through acute plague even though no specific immunity develops.

V. The Alpine Marmot (*A. marmotta*)

These rodents, which in former centuries perhaps played some part in the spread of European plague, must now be classed among the experimentally susceptible ones. They are, however, of considerable interest to us in that they are close relatives of the Siberian marmot and successful hibernation experiments have been performed upon them. Dujardin-Beaumetz and Mosney (16) injected *Bacillus pestis* subcutaneously into three European marmots. One non-hibernating animal died after $2\frac{1}{2}$ days. The others, which hibernated, succumbed after 61 and 115 days respectively, showing no local reaction or buboes, but foci of chronic pneumonia, in which plague bacilli were present in enormous numbers. These French observers consider this as sufficient proof by analogy that the Siberian tarabagan is a "reservoir" for plague, the virus being held in abeyance during the winter. Though this conclusion seems rash at first, it seems to be justified by our latest experiments upon the true Siberian marmot.

VI. The Tarabagan or Siberian Marmot (*A. bobac*).

Introductory remarks. The Siberian marmot, like the susliks of South Russia, undergoes prolonged hibernation. Most authors agree that plague stricken animals do not seek shelter in the burrows, but stay out and die in the fields. Such rodents have been found on the surface long after the onset of the hibernation period, having either not slept at all, or awakening with the development of symptoms.

For these reasons it is doubted by several observers whether the virus is permanently kept alive in the tarabagan. They contend that in autumn a complete separation takes place between healthy animals which retire to the holes, and sick ones which remain outside to die. Various theories are sug-

gested to explain how the tarabagan population becomes again infected could be preserved in the dark and moist burrows of thought that the animals contract the disease by gnawing human plague corpses. This hypothesis is not only intrinsically weak but is disproved by the fact that importation of human plague does not take place at all. It also seems unlikely that infection could be preserved in the dark and moist burrows of the tarabagan and propagated through animals searching for mates or otherwise chancing into such holes. A theory maintained by Sukneff (17) deserves attention. According to this observer the reservoirs of plague are not the tarabagans but certain species of small rodents. Among his arguments are (a) that the tarabagan does not suffer from chronic plague and (b) that only healthy animals hibernate, whereas sick ones remain outside and die off.

For many reasons we have never agreed with Sukneff (18). Though severely sick animals may remain outside, we have been able to show experimentally that those developing the disease hibernate in much the same way as healthy ones. Regarding chronic plague in tarabagans we have supplied evidence of its existence in laboratory animals. The question whether chronic plague exists in naturally infected tarabagans does not seem fully answered. Some of the pathological changes observed by us in 1923 suggested such a condition, but our latest investigations show that it is not always easy to definitely interpret such findings. However, as already mentioned in the foregoing pages, the presence of chronic plague is certainly not a *sine qua non* for the perpetuation of plague in a rodent species. One is tempted to believe that the presence of rodents affected with chronic plague complicates the question as to how the virus is kept alive, since there is evidence for the development of some immunity in this species. Be this as it may, it is easy to explain how plague is propagated among the tarabagans during the *warm* season without their extinction. For these rodents live in families and as a rule do not stray far away from their burrows, but keep within reach so as to escape danger. It seems probable that the disease among them has usually a familial character. Undoubtedly the infection creeps slowly from burrow to burrow and then from settlement to settlement. In this an important rôle is played by very sick animals which stagger about aimlessly and may shed some of their parasites anywhere. These parasites will seek new hosts as their old ones die off. Chance meetings may also take place among animals of different families, during the mating season, in case of danger, etc.

Earlier hibernation experiments. The pivotal point of our problem is therefore the fate of the virus during the long

winter. Dujardin-Beaumetz' and Mosney's experience, suggestive as it is, cannot be taken as a valid proof that *Bacillus pestis* is preserved in the Siberian marmot throughout the hibernating period. Likewise our former winter experiments (1922-1923) upon tarabagans (19), though showing that infected animals may continue to sleep and succumb to infection considerably later than those infected in summer, were not wholly satisfactory. For, in order to obtain as much preliminary experience on hibernation as possible we adopted the following procedures in attacking the problem: (a) different methods of infection were chosen, so that comparisons could be made among small subgroups only; and (b) the animals were daily handled in order to have their temperatures taken. In all probability this often, perhaps inevitably hastened their death.

Plan for new hibernation experiments. To eliminate possible errors, we approached the hibernation problem again, and after careful consideration formulated the following plan:

(a) To extend our program over two winters, and during the first winter (1926-1927) to wait for the spontaneous death of the test animals, handling them as little as possible and killing some of the infected animals at regular intervals during the winter of 1927-1928, in order to study in more detail the manner in which the virus is preserved;

(b) To infect the animals with doses of uniform size by pricking the paw, choosing the inner aspect of the paw of the left hind leg (inoculation by pricking the tail would be more satisfactory in certain respects, but one cannot be so certain of the localization of the buboes);

(c) To infect groups of two tarabagans fortnightly and to infect with each group a guinea pig subcutaneously with the same material in order to confirm its nature and virulence;

(d) To begin the experiments with our strain "V"* and to pass it as far as possible from tarabagan to tarabagan and if this failed to use a culture from the guinea pig of the preceding group;

(e) To house the infected tarabagans in the unheated outbuilding used in our former hibernation experiments.

This plan had to be modified in the course of the work because a few animals died prematurely of unspecified lung or intestinal disease, due perhaps to the extreme cold. Hence after the third experiment only one animal of each group was kept in the outhouse during the winter of 1926-1927, the other

* This is a strain of tarabagan origin, which has since 1923 been repeatedly passed through laboratory animals. It has a high and stable virulence.

in the warm plague room of the laboratory. The evolution of infection did not seem to be affected by this difference in temperature.*

Experiments in the winter of 1926-1927. In the winter of 1926-1927 we infected 9 guinea pigs and 16 tarabagans (14 hibernating). Little need be said about the former. With one exception they all died of acute plague within 3-7 days, mostly on the fifth day and yielded typical cultures. The exception was a guinea pig of the first group which succumbed to plague on the eleventh day. That the initial culture was virulent is proved by the fact that one tarabagan of this group which did not remain sleeping, died after six days of acute plague. A guinea pig infected with material from guinea pig 1 also succumbed after five days. Hence the lengthy survival of guinea pig 1 might have been due to individual resistance. The following is to be said of the 14 tarabagans hibernating at the time of infection:

	Number.	Condition after infection.
a. Died after 2 days, no plague ...	1	Continued to hibernate
b. Died after 5-19 days with signs of manifest plague with bacteremia	6	<div> { Slept fairly well 1 { Sleep interrupted 4 { Awoke 1 </div>
c. Died after 22-60 days, no plague	3	<div> { Continued to hibernate .. 2 { Sleep interrupted 1 </div>
d. Died after 28 and 48 days respectively with signs suggesting residual plague	2	Continued to hibernate
e. Died after 88 and 130 days respectively, i.e. a few days after awakening at the normal end of hibernation with signs of local and bacteremic plague	2	See text

One striking feature of the above summary is the great variation in the results obtained. It seems questionable if the same holds true in nature. We tried to reproduce the undisturbed quietness, low but only slightly oscillating temperature, etc., which reigns whenever the tarabagans sleep in their natural habitat, but were not completely successful. Hence it is more than probable that a certain percentage of our experimental animals died prematurely, while others would have survived under natural conditions. Nevertheless, valuable conclusions can be safely drawn from the experiments. These results may now be discussed in detail.

A. *Animals showing no signs of plague at post mortem (Groups a and c).* Of the four tarabagans composing this

* A table showing the temperatures of the two rooms will be found in the appendix.

group one showed bronchopneumonic foci confirmed by histological examination; two had subpleural petechiae and had presumably succumbed to a lung infection; the fourth, dying two days after infection, displayed signs of an acute enteritis. In none of these animals were any macroscopic changes noted at the site of infection or in the inguinal glands. In one instance a few non-characteristic bacilli were seen in smears from the spleen, while in the pneumonic case such were present in preparations from both the spleen and the lung. All cultures were sterile.

It is possible that an exhaustive experimental and histological examination would have yielded some traces of plague infection, but because of our plan to make a systematic search for such next winter, we thought it wise not to spend too much time and energy upon chance findings. It is therefore difficult at present to draw any final conclusions in regard to this group. However, some of the hibernating tarabagans may escape infection or overcome it.

B. Animals showing signs of local (probably residual) plague (Group d). The findings in two animals under this group point perhaps in the same direction. Their protocols are the following:

Tarabagan 3a. Infected Jan. 3, 1927; died Jan. 31, i.e. after 28 days, having hibernated well throughout. *Post mortem.* Big well nourished animal; no changes visible at site of infection. In left inguinal region one gland hyperemic and increased to size of a half-pea; another of about the same size and filled with caseous matter. Lungs pink; nothing abnormal. Liver somewhat enlarged, dark-brown. Spleen slightly enlarged, but firm. Bladder full. No pelvic bubo. *Bacteriological examination.* Smears from the inguinal glands showed a few suspicious bacteria. Smears from the heart blood showed a few faint bipolar-stained bacilli; those from the spleen doubtful. Cultures from the internal organs negative. A guinea pig infected percutaneously with material from the glands survived. *The histological examination** of the caseating gland showed the capsule much thickened with leucocytic infiltration. Extensive destruction of lymphatic tissue, only remnants of which were to be seen among necrotic masses. *Bacteriological diagnosis* difficult owing to the presence of an enormous mass of granular debris; no definite bacilli seen, but involution forms perhaps present. In the hyperemic gland the capsule was thickened and infiltrated. Lymphatic tissue much engorged; numerous deposits of a brownish pigment. Single bipolar-stained bacilli encountered in the medulla and near the hilus.

* For our histological examinations we used principally hemalum-eosin, but also Kossel's method of staining. Ten c.c. of concentrated aqueous solution of methylene blue (Hoechst) are diluted in 100 c.c. of distilled water. To this is added a 5 per cent. solution of sodium carbonate (cryst). Six c.c. of a 1 per cent. aqueous eosin A extra (Hoechst) are now gently added while shaking, so as to prevent formation of a sediment. The solutions are prepared and filtered immediately before use. Sections are stained in this solution for from 10 to 30 minutes; then differentiated in dilute acetic acid (1 drop glacial acetic acid in Petri dish filled with distilled water) until the pink color appears in parts. They are then passed through 95 per cent., then through absolute alcohol until no more blue color comes out, and finally through xylol and mounted in cedar wood oil.

Tarabagan 5a. Infected Feb. 1, 1927; died March 21, i.e. after 48 days, having hibernated well throughout. *Post mortem.* Large congested area at site of infection. Inguinal glands on left side somewhat enlarged, but not congested. Lungs pink with small hyperemic areas on surface, Spleen not enlarged but slightly softer than normal. Bladder full. *Bacteriological examination.* Cultures from the internal organs sterile. A guinea pig infected subcutaneously with material from site of infection survived. *Histological examination.* No marked congestion around area of local reaction. In the subcutaneous tissue hemorrhages of varying extent noted with slight leucocytic infiltration in places. The latter under high power showed scanty bipolar-stained bacilli. No marked changes in the inguinal glands; possibly trabeculae somewhat enlarged. Capsula of the gland undoubtedly thickened, but no leucocytic infiltration. In the lungs at places were found groups of alveoli filled with a cellular exudate, mostly erythrocytes (red hepatization). No plague bacilli nor other microorganisms.

While there is little doubt that a few plague bacilli still persisted in these tarabagans (one at the site of infection and the other in a regionary lymph gland), it is difficult to ascertain their true significance. We shall later bring evidence to show that plague bacilli introduced during the hibernation period may remain at the site of infection, cause certain changes there, and finally lead to a general infection when the animal awakes. Naturally the question arises whether the findings in the two tarabagans above do not constitute such local deposits of bacteria which would have led to manifest plague, had the animals survived until spring. This possibility will have to be affirmed or disproved by our investigations next winter. At present we are inclined to believe that the changes noted in tarabagans 3a and 5a were possibly the result of a successful struggle with the invaders, and that the few bacilli still remaining would have eventually disappeared, had the animals not died prematurely.

C. Animals dying of manifest plague during the hibernation period (Group b). Only one of the six animals of this group continued to hibernate fairly well after infection; four slept interruptedly, while the sixth was up throughout the six days of its illness. These observations are not easy to explain. There is undoubtedly much reason in the contention of authors like Gaiski that infected hibernating rodents wake up because symptoms of plague develop in them, but we doubt if this always holds true. Possibly in some instances the disease runs a rapid course because the animals are disturbed on account of the artificial conditions in which they are kept. The following table shows the macroscopic findings of this group.

The detailed description of the more important changes may now be given.

1. *Local reaction and bubo.* Even the few tarabagans in this series seem to fall into different groups. Thus we had a

case of purely "septicemic" plague without local changes (Tarabagan 1a), and animals with a somewhat prolonged course of the disease and subacute changes characterized by the presence of suppuration (Tarabagans 4b and 5b).

Tarabagan 7b, succumbing on the 19th day after infection, though belonging to this group, did not show such marked gross changes. It resembled macroscopically the animals 6a and 6b which displayed signs of acute plague, thus standing between the two groups just mentioned.

With exception of Tarabagan 1a (which was not investigated in this respect), positive *bacteriological* results were obtained in each instance from the site of infection and bubo.

The macroscopic findings were generally confirmed by *histological* examination. Tarabagans 6a and 6b showed very acute changes at the site of infection and in the bubo. At the former leucocytic infiltration and hemorrhages were noted in addition to much congestion. Plague bacilli were present in enormous numbers, often forming large clusters and bizarre nets in the areas of infiltration and hemorrhage. The affected lymph glands showed in both cases severe congestion and hemorrhage in the adenoid tissue (in case of 6b also early caseation); plague bacilli were very numerous, again often in big clusters. The capsule of the glands was thickened in places and infiltrated with leucocytes. Periadenitis, not well marked macroscopically, was found by microscopic examination, characterized by much congestion, leucocytic infiltration and even hemorrhage in places.

The local changes in animals 4b and 5b were different from those just described. Hemorrhage was absent and instead of the more diffuse infiltration with leucocytes one found more or less well-defined agglomerations of cells. In tarabagan 4b some reaction was present in the surrounding connective tissue, where in places marked congestion and cell proliferation were noted. Plague bacilli were quite numerous, often forming clusters in the abscess-like formations. In the buboes there was no hemorrhage and less marked congestion than in the preceding cases. Smaller and larger caseating areas were present. Plague bacilli were quite plentiful in the bubo of Tarabagan 5b, where they were arranged mainly in clusters; in 4b they did not appear so numerous and involution forms were encountered. Periadenitis was quite marked in this case though no hemorrhage was noted; the capsule of the gland was apparently involved. In animal 5b, changes round the gland were not conspicuous; some alteration of the capsule was noted.

TABLE II.

Tarabagan number.	Died days after infection.	Local reaction.	Bubo.	Liver.	Spleen.	Lungs.	Other organs.
1a	6	None	None	Fat infiltration. Subcapsular hemorrhages	Slightly enlarged and softer	No marked changes	Hemorrhages in mesenterium. Serosa intestine congested
4b	12	Marked suppuration in places	Size bigger than windsor bean. Caseous matter on section in places	Enlarged congested	Swollen and distinct nodes	Subpleural petechiae	Kidneys congested
5b	10	Abscess size of half-pea	Size over windsor bean. Some suppuration.	Small nodes and widely distributed hemorrhages size of lentil	Much enlarged. Nodes up to size of lentil	Some areas of congestion	Omentum adherent to spleen. Congested. Stomach mucosa congested. Small intestine at places congested. Mesenterial glands enlarged and congested

TABLE II.—Continued.

Tarabagan number.	Died days after infection.	Local reaction.	Bubo.	Liver.	Spleen.	Lungs.	Other organs.
6a	5	Hemorrhages and indistinct infiltration	Small. Inguinal glands of right side enlarged and congested	Congested. Hemorrhages below capsule	Not markedly changed	No marked changes	Hemorrhages in omentum
6b	5	Infiltration size of two peas	Size of two peas. Marked periaadenitis with hemorrhages	Fat-infiltration. Subcapsular hemorrhages	Much enlarged slightly softer	Pale. Fine subpleural hemorrhages	Tubes and ovaries congested
7b	19	Infiltration size of two peas	Size of windson bean. Oedema of abdominal subcutis	No marked changes	No marked changes	Pale	Hemorrhages in omentum and mesenterium

NB. Diagnosis in case 6b confirmed by smears and cultures; in all other instances also by experiment.

That the gross appearances in Tarabagan 7b were not so typical is explained by the fact that in this case hemorrhages and some diffuse leucocytic infiltration were present at the site of infection. But here also one noted under the microscope abscess-like formations of leucocytes. There was a reaction on the part of the connective tissue which tended to encapsulate the abscess-like formations or to penetrate them. Plague bacilli were quite numerous but not often arranged in clusters. In the bubo there was marked congestion and hemorrhages were noted at places in addition to some caseation. Plague bacilli were numerous, occasionally in groups, but on the whole did not seem so plentiful as in the acute cases. The capsule was moderately thickened with some leucocytic infiltration and cell proliferation. The tissues around the gland were congested and showed leucocytic infiltration which was perhaps not so marked as in the animals succumbing quickly.

2. *Liver and spleen.* In every animal of this group positive bacteriological findings were obtained from the liver and the spleen. The morbid changes corresponded in general to those observed in all bacteremias, especially plague; sometimes the absence of marked lesions was conspicuous (see table). Only two cases with peculiar features deserve special discussion.

Tarabagan 4b. The spleen of this animal showed indistinct nodes besides much acute swelling. Histological examination confirmed the presence of large and small areas where lymphocytes and leucocytes, at places mixed with red blood corpuscles, were embedded in uniformly contrast-stained, necrotic tissue. Plague bacilli (usually single) were fairly numerous within such areas. In other areas, especially in the periphery of the necrotic nodes, they were seen in enormous numbers, forming clusters and nets. Even within the Malpighian bodies some organisms were encountered, especially at spots where a little hemorrhage seemed to have taken place.

Tarabagan 5b. Here marked appearances of "nodose" plague were noted in both the liver and the spleen. Histological investigation revealed marked alterations in the former, consisting of a marked congestion and infiltration of the liver cells with fat globules; in some places more or less extensive hemorrhage was seen, while in others the liver tissue was more or less destroyed; leucocytes alone or mixed with red corpuscles abounded in the damaged tissue. Plague bacilli were found in moderately numerous clusters, often situated at the periphery of the necrotic areas. Similar but larger nodes were encountered in the spleen, bacilli in groups occurring near their circumference.

Tarabagans suffering from such "nodose" plague have been recorded in the past. Thus:

First, a few animals, including one shot in the fields by Barykin in the year of 1907 (20), showed small greyish nodes in the spleen;

Second, one naturally infected animal found by Sukneff in 1923 had some bulging nodes in the lungs, many nodules and hemorrhagic spots in the liver; the spleen of this animal having been partly eaten by eagles, its condition could not be ascertained (19), but histologically the lesions in the liver, although further advanced, were similar to those observed in Tarabagan 5b (21), and;

Third, one tarabagan infected conjunctivally in the course of our former winter experiments (1922-1923) and succumbing on the 17th day after infection showed numerous pin-head yellowish-white nodules in the liver and larger white nodes (size of a lentil) in the spleen.

Cultures from the second and third cases were somewhat impaired in virulence. For this and other reasons we were inclined to consider such nodose changes as the result of a subacute or even chronic stage of the disease. As shown by our recent experience, such alterations may develop comparatively quickly, so that one must be chary of hasty conclusions. We believe that in the tarabagan as well as in other rodents such types as acute, subacute and chronic plague ought not to be separated by any sharp, arbitrary lines. In all probability graduations between these types may also exist.

II. Animals succumbing to plague after awakening from hibernation (Group e). The two animals belonging to this group may now be described:

1. *Tarabagan 3b*, infected on January 3, 1927, was up to the 10th kept in the outhouse, later in the laboratory stable. It slept well with short intermissions up to March 8th. From that time it was usually drowsy although eating a little food occasionally. When disturbed it did not bark, was not shy of human beings, and generally remained listless. When occasionally taken out of the cage, it neither resisted nor attempted to run away; sometimes the hind legs looked paralyzed. On the whole it gave the impression of suffering from a chronic disease (plague?) rather than being in a state of hibernation. That the latter condition prevailed became evident early in May (2d) when the animal began to react better, displaying its teeth when approached, but not barking. A week later (May 9th) it was wide-awake, feeding greedily when a carrot was thrown in the cage. This condition remained the same up to the morning of May 12th, when it was last seen alive. Next

morning (May 13th) it was found dead, i.e. 130 days after infection.

Post mortem. Little fat, no emaciation. No marked local reaction. Small but markedly congested bubo in left inguinal region; some hemorrhages in the adjoining fascia. Right inguinal glands slightly enlarged and congested; cervical glands somewhat congested but not enlarged. Lungs oedematous; right lung showed large areas of congestion. Numerous petechiae on epicardium. Liver not enlarged, brownish-yellow in colour; some perihepatitis in form of white linear thickening of the tissue. Spleen softer than normal but not enlarged; congestion in places. Retroperitoneal hemorrhages. No other conspicuous changes. *Bacteriological examination.* Smears from bubo, heart, lung and spleen positive for plague bacilli. Cultures from bubo gave no growth; from heart, lung, liver and spleen positive but contaminated. Two guinea pigs infected immediately at *post mortem* succumbed to plague. The first which was pricked with a needle dipped into the bubo, died on the third day, while the second, rubbed on the shaved skin with material from the bubo, heart and lung, succumbed on the fourth day.

Histological examination. Sections from the bubo showed as far as the gland tissue was concerned appearances similar to those in the acute cases. Severe hemorrhage was present, leading at places to a disintegration of the gland structure. Plague bacilli were very numerous, often arranged in big clusters at the periphery of the gland. The capsule, however, showed at places marked thickening and cell proliferation, but leucocytic infiltration was absent. The tissues near the bubo were much congested; hemorrhages were occasionally met with, but no leucocytic infiltration.

A slightly enlarged *lymph gland from the right groin* showed much congestion but no hemorrhage. Plague bacilli were numerous in the larger blood vessels, but scarce outside of them. The capsule was not perceptibly changed. The surrounding connective tissue was less congested than in the case of the bubo, no leucocytic infiltration could be seen, only small hemorrhages. *The liver* showed at places thickening of the capsule. Congestion and parenchymatous degeneration were present. Fairly numerous plague bacilli were noted in both vessels and capillaries, sometimes in small clusters. *The spleen* was much congested. The Malpighian bodies were comparatively small, the trabeculae prominent. Plague bacilli occurred in large numbers but were more evenly distributed than in the foregoing cases, so that no big clusters were met with. *The lungs* were congested and showed foci of broncho-pneumonia; the exudate was mostly cellular, red blood corpuscles being more numerous than white ones. Plague bacilli were plentiful, rarely in groups. *The kidneys* showed congestion and parenchymatous degeneration, plague bacilli being noted within the vessels only. In the *retroperitoneal tissue* there were large hemorrhages; here the bacilli were usually grouped together in small clusters or loose nets.

2. *Tarabagan 4a* was infected on January 17th, 1927, and kept throughout in the outhouse. It slept well with almost no interruption up to the beginning of April. Occasionally up from April 4th the animal was wide-awake on April 11th; on this day it was seen to sit on the straw in its cage but to hide itself immediately when approached. It was well and feeding up to April 13th, when seen for the last time before death. It

was found dead on the morning of April 15th, i.e. 88 days after infection.

Post mortem. Medium sized animal, still moderately fat. Some reaction noted at site of infection (superficial layer of musculature) where congestion and perhaps some infiltration were present. *Left inguinal glands* slightly enlarged but not markedly congested. *Lungs* oedematous, anterior parts pale; areas of congestion in dorsal parts of both lower lobes. *Liver* congested showing some indistinct sub-capsular hemorrhages. Some perihepatitis over left lobe in form of a net of white, thickened tissue. *Spleen* not markedly changed. *Stomach* showed petechiae below mucosa; full of bile-stained liquid. *Duodenum* and upper part of *jejunum* much congested; their contents bloody. Subserous hemorrhages on duodenum.

Bacteriological examination. Smears from the spleen showed numerous bipolar-stained gram-negative bacilli. In films from bloody intestinal contents bacilli similar to *Bacillus coli* were present in addition to large gram-positive bacilli. Cultures from heart, liver and spleen were typically positive, those from the lung somewhat atypical, but suspicious. Culture from intestine was negative for *Bacillus pestis*. Altogether five guinea pigs were inoculated. One received a dose of culture from the intestine upon the shaved skin and survived; the others all succumbed to plague; the first pricked with material from the site of infection died on the third day; the second rubbed with material from the lung and intestine died on the seventh day; the third rubbed with liver culture succumbed also on the seventh day and the fourth rubbed with lung culture on the ninth day.

Histological examination. After a prolonged search a small abscess was found at the *site of infection*. The cells at the periphery stained fairly well; those in the center had undergone necrotic changes, so that a diffusely stained mass was present showing at places indistinct nuclei. The connective tissue near the abscess was markedly changed; rich in cells and blood vessels it had the aspect and arrangement of granulation tissue. Small hemorrhages were seen in places but no agglomerations of leucocytes. No bacilli could be demonstrated in the center of the abscess, though they were fairly numerous at the margin, occurring both in typical and involution forms, the former occasionally in clusters. About the abscess plague bacilli were plentiful in the vessels, but scarce in the tissue, with the exception of the hemorrhagic areas; even here no large clusters of bacteria were seen.

One enlarged left inguinal gland showed under the microscope some congestion and small hemorrhages. The connective tissue was increased in volume at the cost of the parenchyma. The arrangement of the bacilli resembled that in the connective tissue at the site of infection. The capsule of the gland was thickened with cell proliferation in places. Some congestion was present around the gland but no marked periadenitis. The capsule of the liver was thickened; cell proliferation was noticeable. Marked congestion was present throughout the organ with occasional hemorrhages. The liver cells showed signs of fatty degeneration. Plague bacilli were plentiful in the vessels and capillaries; at and near the hemorrhages they also occurred outside the vessels, now and then forming clusters.

The *spleen* was very rich in blood. The capsule was somewhat thickened and showed signs of cell proliferation. The Malpighian bodies were small and the follicles prominent, but this was not so conspicuous as in the foregoing case. Plague bacilli were very numerous but big clusters were absent. The lungs showed general congestion and foci of broncho-pneumonia especially near the surface. Here the

alveoli were filled with a serous, less often a cellular exudate; in the latter red corpuscles prevailed. Bacilli were moderately numerous in the foci, often in small groups; in other parts of the lung they were present in the vessels and capillaries only.

The *kidneys* were congested and showed parenchymatous degeneration. Bacilli were quite numerous in vessels and capillaries. Sections from the stomach showed extensive submucous hemorrhage containing bacilli in groups. The wall of the small intestine was much congested with hemorrhages, in places reaching the surface of the mucosa. Big clusters of bacilli occurred in and around the hemorrhages.

It can thus be seen that two of our tarabagans infected while sleeping, continued to hibernate. They succumbed to plague with bacteremia in the spring, after they had been up and apparently well for a few days; in one of those the hibernation period appeared to be prolonged.*

The bacteria present at the time of death in the blood and organs of one animal (tarabagan 4a) were little impaired in virulence, if at all; in the other they may possibly have had an increased virulence.

The question of the area in which the plague bacilli are preserved in such animals until the disease becomes manifest, is not yet fully answered. There is little doubt but that in Tarabagan 4a, they remained at the site of infection. In Tarabagan 3b, however, no local reaction could be detected, and, though some older change might have been present in the inguinal glands, we cannot affirm that the bacilli reached them at an early stage of the infection. Some chronic lesions were noticed in the liver and in the spleen of both animals, but it is difficult to gauge their significance. Possibly they were caused by toxins circulating in the blood. We hope to elucidate all these questions during the next winter.

VII. Summary And Conclusions

Evaluating the results of our latest investigations, we must admit that the two animals surviving up to spring had contracted infection *during and not before* the onset of hibernation, while the twelve others died during the winter, six of acute and sub-acute plague. As stated above, conditions are certainly much more favorable in nature than under artificial laboratory conditions. It seems improbable that the percentage of rapidly evolving plague cases can be as high as we have witnessed in our experiments. It is also probable that in some of the naturally infected animals plague does not develop at all or remains localized, followed by recovery. However this may be, there is little

* It is hardly necessary to mention that an accidental infection of the animals during their observation is out of the question. Our tarabagans kept at Harbin since the summer, 1926, were free from parasites. No experiments with rats or other flea-bearing animals were performed throughout the whole time. The few infected guinea pigs in the laboratory stable were placed in buckets away from the tarabagans. The latter were kept in individual cages and every possible precaution was taken against the possibility of infection through food or from other sources.

H. APPENDIX.

1. Tabulation of temperatures

(a) in the unheated out-house, (b) in the basement of the laboratory, where the animals of the series (winter 1926-1927) were kept.

Date.	Temperature in centigrade.		Date.	Temperature in centigrade.	
	(a)	(b)		(a)	(b)
Dec. 2.....	— 7	—	Feb. 19.....	—10	—
„ 3.....	— 7	—	„ 21.....	— 9	—
„ 6.....	— 9	—	„ 22.....	— 9	—
„ 8.....	—10	—	„ 24.....	—10	11
„ 9.....	—12	—	„ 26.....	—9 Mean:	— Mean:
„ 11.....	—12	—	„ 28.....	—10 —11.25	10 +10.9
„ 13.....	—12	—			
„ 15.....	—13	—	Mar. 4.....	— 9	12
„ 18.....	—13	—	„ 5.....	—	10
„ 20.....	—12	—	„ 7.....	— 8	13
„ 22.....	—11	—	„ 9.....	— 8	16
„ 24.....	—12.5	—	„ 11.....	— 7	—
„ 26.....	—13	—	„ 14.....	— 7	11
„ 28.....	—14 Mean:	—	„ 16.....	— 8	10
„ 30.....	—12 — 11.3	—	„ 18.....	— 6	11
		—	„ 19.....	— 6	—
Jan. 2.....	—10	—	„ 21.....	— 4	—
„ 3.....	— 9	—	„ 23.....	— 3	11
„ 4.....	— 8	—	„ 25.....	— 1	12
„ 6.....	— 8	—	„ 28.....	1 Mean:	12 Mean:
„ 8.....	— 6	—	„ 30.....	4 —4.8	— +11.8
„ 10.....	— 8	—			
„ 12.....	— 9	—	Apr. 1.....	4	—
„ 14.....	—12	—	„ 4.....	2	12.5
„ 17.....	—16	—	„ 6.....	2 Mean:	—
„ 19.....	—16	—	„ 13.....	4 +4.0	11.5
„ 21.....	—17	—	„ 19.....		13
„ 24.....	—17	—	„ 20.....		11
„ 26.....	—16	—	„ 21.....		15
„ 28.....	—16 Mean:	—	„ 22.....		12
„ 31.....	—15 — 12.5	12	„ 23.....		14
			„ 25.....		12
Feb. 3.....	—14	—	„ 28.....		13.5
„ 5.....	—13	11	„ 29.....		15 Mean:
„ 7.....	—13	12	„ 30.....		15 +13.1
„ 8.....	—	10.5			
„ 9.....	—14	—	May 3.....		12
„ 10.....	—	11	„ 4.....		13
„ 11.....	—13	—	„ 5.....		15
„ 14.....	—11	11	„ 9.....		14 Mean:
„ 18.....	—	10.5	„ 11.....		14 +13.6

doubt that the tarabagan fleas, like those of the susliks, play an important part in spreading the disease in winter as well as in summer, and are able to preserve the virus, especially during the cold season, for lengthy periods of time. Supplementing the knowledge attained by our experiments with the above considerations, we can see how plague is propagated among the tarabagans from year to year.

Summarizing the knowledge obtained from the tarabagan with that of wild rodents elsewhere, and comparing the results with facts established in regard to the domestic rats, we may state the following conclusions:

1. The occurrence of rodent plague with bacteremia is a *sine qua non* for the propagation of the disease in the wild as well as in the domestic species;

2. Cases with chronic plague (in the strict sense) do not play any important rôle in the preservation of the virus;

3. Besides cases of acute and subacute plague, carriers with bacteremia may occur; their significance is not yet fully established;

4. The hibernation period to which some of the wild rodents suffering from natural plague are subjected, is not a hindrance to the perpetuation of the disease, but on the contrary an indispensable link for the preservation of both the virus and the species.

In order not to confound the issues, no mention has been made in the text regarding migration. This may be important in two ways; first, by *immigration* of healthy animals into an infected locality; and second, by *emigration* of infected animals or carriers of infected fleas into a healthy region. It is evident that in the first instance an impetus would be given to any enzootic present, while in the second ample fuel would be provided for the virus regardless of its fate at the place of origin.

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THE PERPETUATION OF PLAGUE AMONG WILD RODENTS, WITH SPECIAL REFERENCE TO THE SIBERIAN MARMOT

(SECOND COMMUNICATION)

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Table Of Contents

- A. Introduction.
- B. Tarabagan dying at an early stage of the disease.
- C. Tarabagans succumbing to acute plague.
- D. Tarabagans showing residual plague.
- E. Tarabagans showing latent plague.
- F. Discussion of latent plague.
- G. Summary and conclusions.

A. INTRODUCTION

In a previous paper (The Perpetuation of Plague among Wild Rodents) we described the first stages of a new series of plague experiments upon hibernating tarabagans commencing 1926-7. Of the 14 animals then infected six succumbed more or less quickly to manifest plague. Four demonstrated at autopsy no trace of infection; these were however, not exhaustively examined. Two of our test animals showed postmortem lesions suggestive of residual plague, while the remaining two, succumbing 88 and 130 days respectively after inoculation, i.e. a few days after awakening at the normal end of hibernation, displayed signs of generalised plague together with older changes at (a) the inoculated site and (b) the regional lymph glands.

Satisfactory as this evidence appeared, we had realised from the beginning that a second set of observations would be desirable in which some at least of the test animals should be killed at varying intervals so as to ascertain from another viewpoint how the virus is preserved. We have now to report upon this second series of experiments.

During the winter of 1927-28 we inoculated altogether twenty tarabagans, using the same technique and the same strain as in the year before. Certain animals succumbed spontaneously

to plague or died of intercurrent disease (with some evidence of plague). The rest were killed at various intervals after infection. Our results are summarised as follows:—

TABLE No. I.

P.M. findings:	Died spon- taneously:	Were killed:	Total:	Remarks:
Early signs of plague infection in addition to unspecific pneumonia.	Tb. 21.	—	1	Died 3 days after inoculation.
Manifest plague with bacteremia	Tbs. 12, 13, 14, 20, 22, 23 27, 28.	—	8	Died 5-8 days after inoculation.
Residual plague.	—	Tbs. 11, 29.	2	Killed after 21 and 118 days respectively.
Latent plague.	Tbs. 17, 24	Tbs. 15, 16, 18, 19, 25, 26, 30.	9	Tbs. 17 and 24 died after 12 and 14 days respectively, while the others were killed 16-40 days after inoculation.
				Grand Total....20

The above animals fall under 4 groups, which may now be discussed *seriatim*.

B. TB. 21, DYING AT AN EARLY STAGE OF THE DISEASE

This animal was infected on January 21, 1928, together with three other tarabagans which all succumbed within 5 days to acute generalised plague. Tb. 21, which slept well at the time of infection was found dead on the morning of January 24. Post mortem findings were as follows:

Fairly big, female animal.

At the site of infection a large area of reaction in which hemorrhagic districts were interspersed with non-hemorrhagic ones; small abscesses seemed present in the latter. Microscopically one noted in the connective tissue between the muscle bundles as well as in the surrounding fascia hemorrhages and agglomeration of leucocytes which at places became so dense as to form small abscesses. Many gram-negative plague-like bacilli were seen within the abscesses, when they often formed clusters and small nets; round the abscesses bacilli were generally scanty except at some spots containing dense leucocytic infiltrations.

One of the regional inguinal glands was enlarged and hyperemic. Microscopically its capsule showed thickening and

perhaps some cell proliferation in parts. Varying hemorrhages were noted within the gland while the surrounding tissues were congested. Only a few gram-negative, plague-like bacilli were noted within the gland.

The liver was markedly congested at autopsy; one indistinct node was noted. Microscopically distinct congestion was found together with numerous hemorrhages. In addition leucocytic agglomerations (both large and small) were met with. The former, either round or irregular in shape, contained a few areas with scattered leucocytic infiltration, the cells being evidently embedded in a necrotic ground substance. The leucocytes composing the larger nodes were partly eosinophil.¹ Some atypical, though suspicious gram-negative bacilli, were noted in the leucocytic infiltrations; away from these there were less.

The spleen apparently not much changed at post mortem was histologically found to be moderately rich in blood with fairly large follicles and prominent trabeculae. In Kossel-and gram-stained specimens a few bacilli similar to those in the liver were found.

The lungs appeared very much congested. The microscope revealed also the presence of foci of confluent broncho-pneumonia with a cellular exudate in which red blood corpuscles greatly outnumbered the white ones. Very few gram-negative suspicious bacilli were seen in the peribronchial glands and alveolar walls, while occasionally small groups of gram-positive cocci similar to staphylococci were encountered in and near the pneumonic areas.

The kidneys presented no unusual macroscopic features. Histologically moderate congestion was noted in addition to some parenchymatous degeneration. Numerous small hemorrhages were seen in the medulla. Very few gram-negative, suspicious bacilli were found in and near these hemorrhages as well as in the glomerular capillaries.

We have no doubt that Tb. 21 died of acute pneumonia. Similar lesions have been noted by us during the past years among some of our infected as well as healthy tarabagans, which succumbed during hibernation. It seems that such animals—though resistant in other respects—are specially liable to contract acute lung inflammation upon the slightest exposure. This is not surprising if we consider how well these marmots, when hibernating under natural conditions, are protected against any abrupt oscillations of temperature.

Some colonies of *staphylococcus albus* were cultivated from the heart, liver and spleen, but none from the lung. Plague bacilli could not be demonstrated with certainty in the cultures

¹ As will be discussed later on, these large nodes are in all probability not due to plague infection but to a parasitic invasion of the liver.

which, however, were taken from the internal organs only. That the animal was in an early stage of plague infection is amply proved by the local findings. In the bubo and some of the internal organs, notably the kidneys, changes were seen somewhat characteristic of this disease. Though a manifest bacteremia was absent, a few plague-like germs were found everywhere. It is difficult to decide whether these were the first harbingers of a general fatal infection or whether the animal, had it survived, would have kept the invaders at bay.

C. TARABAGANS SUCCUMBING QUICKLY TO ACUTE PLAGUE

In addition to complete autopsies and cultural tests in this group proving that all eight animals had succumbed to bubonic plague with secondary septicemia, we resorted to histological examinations as well. Our main object in doing so was to prove to our own satisfaction that our methods of fixing, embedding, cutting and staining the material would not hinder the demonstration of the *B. pestis* in the tissues. The results seen in these manifestly infected animals thus served as controls for the tarabagans of the other groups which were investigated simultaneously. There was *a priori* little hope that the histological examination might materially supplement the knowledge gathered in 1926-7 upon the animals succumbing to manifest plague. For all the tarabagans under discussion at present died rather quickly (within 5-8 days after infection). On the other hand, perhaps due to the unusually mild winter with intervening warm spells, many of the animals were likely to show more advanced changes than the corresponding ones which succumbed quickly in 1926-7.

We proceed now to a more detailed description:

1. *Local reaction.*

All eight animals showed a well-marked local reaction, more or less extended suppuration being macroscopically visible in a majority of the cases. Histologically more or less well defined abscess-like agglomerations of leucocytes were found in every case. In three animals (Tbs. 14, 23 and 27) these abscesses were not prominent as compared with the diffuse leucocytic infiltration around them, while in two (Tbs. 13 & 28) such diffuse infiltration was well marked in addition to the presence of clearly defined abscesses. In the three remaining tarabagans (Nos. 12, 20, 22) only slight or spot-like infiltration was found in the vicinity of the abscesses. More or less marked congestion of the surrounding tissues was the rule while hemorrhages, situated either around the abscesses or in addition some distance from them, were noted in practically all cases. Reparatory changes, as noted in some of the 1926-27 cases, were not evident.

Plague bacilli were in most cases numerous in the abscesses, but as a rule not uniformly distributed, being usually grouped together at the periphery of the leucocytic masses while scanty or even almost absent in the center. Round the abscesses such a clustering of the bacilli was the rule. Here they were most numerous within the diffuse leucocytic infiltrations, sometimes near the abscesses or within hemorrhages or occasionally near blood vessels (? in lymph-spaces). Within the blood vessels they were plentiful in two cases only (Tb. 12 & 13), moderately numerous in Nos. 14 & 20, while in the four other animals they were either scattered or practically absent.

2. *Bubo*.

The presence of a left inguinal bubo was in each instance proved by macroscopical examination. The glands in question, though as a rule but slightly enlarged, were more or less congested. Periadenitic changes, the presence of which was constantly seen under the microscope, were usually noticeable with the naked eye. In the bubo of Tb. 28 a caseating center was noticed at autopsy.

By an unfortunate mistake in the case of Tb. 23 a second piece of the local reaction was preserved instead of the bubo. Thus only seven of the buboes could be subjected to a histological examination. This showed either marked congestion or more or less advanced hemorrhage in the glands. Caseation was found in half the cases; this was conspicuous in Tbs. 22 & 28. The capsule was usually involved, being at places thickened and infiltrated with leucocytes; in Tb. 14, which succumbed 8 days after infection, some cell proliferation was present as well. The changes round the glands usually consisted of hyperemia and spotwise leucocytic infiltration; sometimes hemorrhages were noted.

Plague bacilli were always plentiful within the buboes, often evenly distributed. Round the glands they were concentrated at places only, chiefly in the leucocytic infiltrations. Within the blood vessels bacilli abounded in three instances (Tbs. 13, 14 & 22), while in the others they were more or less scanty.

3. *Liver*.

The liver was in the majority of cases dark in colour; three showed a much lighter, yellowish hue. In Tb. 12 subcapsular hemorrhages could be noted, while Tb. 27 showed irregular, dark spots suspicious of hemorrhage throughout the organ. Tb. 28 had at places thickening of the liver capsule as confirmed by microscopical examination; no marked proliferation of cells could be noted.

Histological examination was undertaken in five cases. In the two instances where the liver had appeared hyperemic at

autopsy, some hyperemia was found microscopically in addition to hemorrhage which not rarely led to destruction of limited areas of the liver tissue. In two of the three cases, which at autopsy suggested fatty changes, fatty infiltration was found under the microscope, while in Tb. 27 only parenchymatous degeneration seemed present besides plentiful hemorrhages. In most of the histologically examined material small areas of necrosis were noted. In such areas erythrocytes were not rarely present, sometimes quite plentiful. Leucocytes which were absent or rare within these foci could occasionally be found in small groups next to them. Such leucocytic agglomerations were commonly met with in apparently unchanged parts of the parenchyma. In Tbs. 13 & 14 evidence of *parasitic* invasion of the liver was found; a more detailed description of these changes will be given in a following paragraph.

In Tbs. 12, 13 & 14 bacteria were plentiful throughout the organ; they formed clusters at the periphery of the necrotic foci, but were less numerous in the center. In Tbs. 27 & 28 plague bacilli were quite plentiful in the parenchyma occurring almost all in the form of small clusters. Here only few bacilli could be detected within the larger blood vessels.

4. *Spleen.*

The spleen in all cases showed at autopsy acute swelling. Microscopically the organ was usually found rich in blood; hemorrhages were present in all five cases investigated. Tbs. 12, 13 & 14 showed in addition small necrotic foci which sometimes were surrounded by hemorrhagic zones. Plague bacilli were plentiful in these cases, forming large clusters at the periphery of the necrotic areas. In Tbs. 27 & 28 no evidence of necrosis was found. Bacilli were here not so numerous or so evenly distributed as in the foregoing cases, being mainly clustered together in small groups.

5. *Lungs.*

The lungs showed at autopsy as a rule some congested areas.

In the five cases examined histologically, marked congestion was found in three (Tbs. 12, 13 and 14), while in two (Tbs. 27 and 28) this was less conspicuous. Bronchopneumonic foci of various sizes were found in every instance; sometimes these seemed to be situated mainly beneath the pleura. The exudate was partly fibrinous, partly cellular or merely cellular, the latter consisting of both leucocytes and erythrocytes in varying proportions.

In Tbs. 12, 13 and 14 fairly numerous bacteria were found in the vessels and capillaries as well as in the bronchopneumonic foci; within the fibrinous exudate they were as a rule absent,

while groups of plague bacilli were seen occasionally around. In Tb. 27 the *B. pestis* were less numerous in the vessels, not evenly distributed throughout the foci, but occurred in clusters. In Tb. 28 the vessels seemed almost free of bacteria; in the foci only a few organisms were seen.

6. *Kidneys.*

The kidneys showed no marked changes at *post mortem* except slight signs of congestion and degeneration.

In the five cases which were further examined, parenchymatous degeneration was present as a rule. Congestion was usually much marked. Hemorrhages were invariably noted in the medulla. Rarely a little hemorrhage was noted in the cortex as well.

Bacteriologically numerous bacilli were found in Tbs. 12, 13 & 14 in the vessels and capillaries as well as in the hemorrhages, while in animals No. 27 & 28 their number was limited everywhere.

7. *Intestine and mesenterium.*

Marked changes were noted in Tb. 28 only. Here a part of the small intestine was found to be much congested; large hemorrhages were seen beneath the serosa and a big mesenterial bubo seemed present.

Histological investigation showed the intestine to be filled with a fibrino-hemorrhagic exudate. The intestinal walls appeared much congested with hemorrhages in all layers.

Several gram-negative bacilli were found in the intestinal contents; the majority like *B. pestis*. Numerous gram-negative and plague-like bacilli were found in the intestinal walls as well, where they were often grouped together.

The mesenterial gland was much changed, in addition to congestion small foci of necrosis being present. The capsule appeared at places thickened and infiltrated with leucocytes. In the surrounding tissue there was congestion and at places also leucocytic agglomeration.

Plague bacilli were found in great numbers in the gland as well as in the infiltrated parts of the surrounding tissues and in some of the vessels. The bacilli were as a rule not evenly distributed, but clustered together.

It can thus be seen that—apart from the unusual feature of a specific intestinal affection in Tb. 28—the macroscopic and histological results obtained in the animals now under discussion were more or less uniform. The only striking difference lies in the quantity and distribution of the bacteria, according to which they may be divided in two groups. In one bacteria were numerous everywhere and as a rule rather evenly distributed. In the other they were on the whole less numerous and more grouped

together in the tissues, while in the blood vessels they were as a rule less numerous or even scanty. It is perhaps not wrong to assume that the animals of the first group were absolutely overwhelmed by the infection, while in the others some attempt to cope with the invaders by localising them may be noticed.

D. TARABAGANS SHOWING RESIDUAL PLAGUE

We think it best to deal now with Tbs. 11 & 29 which—though showing lesions different from those in the animals suspected in 1926-7 to suffer from residual plague—yet evidently belong to the same category.

Tb. 11 was infected simultaneously with Tb. 12 on November 25, 1927. While the latter succumbed after 6 days to acute bubonic plague with general septicemia, Tb. 11 continued to live and was finally killed on December 16, i.e. 21 days after infection.

The results of the autopsy were as follows:

At the site of infection a large abscess was present in the musculature. In its center caseation could be noted in addition to necrosis of the enclosed muscle fibers. The musculature and connective tissue round the abscess showed congestion, moderate infiltration with round cells and cell proliferation. Bacteriological diagnosis was not quite easy owing to the presence of plentiful debris. Undoubtedly, only a small number of atypically short, gram-negative bacilli were present in the caseating center of the abscess, while they were almost absent in the outer layer and only scanty in the surrounding tissues.

A bubo surrounded by fatty tissue seemed present at autopsy, the piece in question being removed and preserved *in toto*. Unfortunately after fixation no glands could be detected in it; apparently the regional glands were so little changed as to escape notice at *post mortem*. The other external lymphatic glands appeared normal.

The liver was congested and showed a few nodes of yellowish-white colour and the size of millet grains. Microscopical examination confirmed the presence of congestion. Smaller leucocytic infiltrations and agglomerations were noted in addition to larger nodes. The latter were round in shape and showed in their center uniformly stained necrotic tissue pervaded by round cells which were especially plentiful at the periphery of the necrotic masses. A thick layer of connective tissue was seen to surround the nodes outside of which a ring of liver tissue was noted which seemed to have undergone fatty degeneration; here as well as in the normal tissue round the nodes small leucocytic agglomerations were present at places, while they were rare in the connective tissue. Some atypical suspicious bacteria were found in the necrotic areas. In the tissue round them they were met with especially in and near the leucocytic agglomerations. A

few suspicious bacteria were present also in the small leucocytic infiltrations and agglomerations while they were very rare in the parenchyma.

The spleen showed several slightly prominent nodes analogous to those in the liver. Microscopically they were also seen to possess a similar structure; necrosis was present in their centers, leucocytes being embedded in the uniformly stained masses and specially gathered together at their periphery. The nodes were surrounded by thick layers of connective tissue; around this hemorrhage was frequently noted. A few suspicious bacteria were seen in the central parts of the nodes; very few atypically big bacteria were met with in the surrounding connective tissue. In the parenchyma suspicious bacilli were present in small numbers.

The lungs were congested and showed several nodes of a similar appearance as those described above. Microscopically the organ appeared much congested. At places small groups of alveoli were filled with an exudate consisting of erythrocytes and leucocytes. In the center of the large round nodes leucocytes were so densely massed that it was impossible to distinguish with certainty a necrotic ground substance. Towards the periphery small and large areas with less dense infiltration were seen where necrosis of the lung tissue was manifest. The nodes were surrounded by thick layers of connective tissue which were—at places rather densely—infiltrated with round cells. Some suspicious bacilli were found in the nodes and very few in the connective tissue round them, the alveolar exudate and the peribronchial glands.

The kidneys showed no marked changes at autopsy. Microscopically there was congestion. Areas similar to those found in the tarabagans of the preceding series (C) were seen in the medulla showing in addition to congestion and occasional hemorrhage formation of connective tissue. Only a few atypical suspicious bacteria were seen, mainly confined to the changed areas of the medulla and the glomerular capillaries, rarely at other places (e.g. in lumina of tubuli).

Smears from the site of infection showed a few suspicious bacteria. Cultures from the internal organs remained sterile. Two guinea-pigs were inoculated subcutaneously, one with an emulsion of two loopfuls from the local pus, the other with a suspension of two loopfuls of heart blood. Both animals survived for more than a month when—being reinfected with virulent plague strains—they succumbed quickly.

Summarising our knowledge obtained from the above tarabagan, we may say that—as far as the gross appearances are concerned—this case should be classed as one of “nodose” plague. It will be remembered that in the previous paper we dealt with

this form saying that in the tarabagan (as well as in other rodents) the presence of nodes does not necessarily indicate a subacute or chronic stage of the disease. However they may point to such varieties, as has been seen in Tb. 11. In this animal the endeavours to grapple with the infection and to encapsulate the foci can be clearly seen. And—contrary to the tarabagans with “nodose” plague referred to last year—only few bacilli (partly involution forms) were found (a) at the site of infection, (b) in the nodes as well as (c) in the organs in general. Comparing these findings together with the negative cultural and experimental results, there seems little doubt that the animal, if not prematurely killed, would have recovered from the infection showing later on signs not of *resolving* but of *resolved* plague.

A more advanced stage was evidently reached by Tb. 29, which was inoculated on February 11, 1928, together with Tbs. 28 and 30, one of which died in 6 days of acute generalised plague. Tb. 29 slept uninterruptedly up to March 21, after which date it was occasionally seen to stir slightly. From about middle of April it was up at times and then partook of a little food. It took the animal about a month to become fully awake. After that time (middle of May) we at first thought it to be ill because it never barked or resisted when approached. No further symptoms developed, however, and finally we resolved to kill the animal on June 8, i.e. 118 days after infection.

The principal findings at autopsy were as follows:

Tb. 29. No changes were detected at the site of infection except some indistinct thickenings of the subcutis. Histologically cell proliferation could be noted in parts of the subcutaneous tissue, and after prolonged searching a small but dense agglomeration of round cells was detected at one place in the immediate vicinity of an artery. In gram-stained preparations small groups of gram-positive cocci were observed in the horny layers over the skin. In the leucocytic area as well as in the subcutis a few gram-negative suspicious bacilli were found; rarely some gram-positive cocci in pairs also were present.

The regional as well as the other external lymph glands were macroscopically found to be slightly enlarged but otherwise unchanged. Some capsular thickening was noted in the inguinal glands only. Everywhere a little brownish pigment was found in the otherwise normal parenchyma. Gram-negative bacilli were scanty in the regional lymph glands, and were only after much search detected in the other glands as well.

The liver was dark-brown in colour showing some mottled capsular thickening, and at one place three small nodes were present together. Under the microscope no marked congestion was noted. The capsule was at places diffusely thickened without

marked cell proliferation. Smaller leucocytic infiltrations were frequently noted; sometimes a thin layer of round cells was seen lying immediately beneath the capsule, particularly at places where the latter was thickened. In addition, large leucocytic nodes, some round and some irregularly-shaped were seen. Within these, areas with necrotic changes similar to those described in the case of Tb. 21 were met with. Eosinophils, though occurring, were not so plentiful as in Tb. 21; a few giant cells were occasionally observed in the center of the nodes. Few suspicious bacteria were found in and near the leucocytic agglomerations.

The spleen appeared both macro—and microscopically free though it contained a few suspicious bacilli.

The lungs were pale except some areas where evidently recent aspiration of blood had taken place. Histological examination showed, besides the presence of districts where the alveoli were filled with fresh blood, other areas where small groups of alveoli were filled with both erythro—and leucocytes. A few gram-negative suspicious bacteria were noted in the lymphatic tissue round the small bronchi, occasionally also in the alveolar walls. Now and then a few gram-positive cocci similar to *Diplococcus pneumoniae* were seen within the alveoli.

The kidneys appeared unchanged at autopsy. Microscopically, in addition to some congestion small leucocytic agglomerations were detected in the cortex, rarely in the medulla. A few atypical gram-negative bacilli were found in and around the agglomerations as well as in the glomerular capillaries.

Smears from the site of infection, the regional glands and the spleen were negative; cultures from the internal organs generally proved sterile; that from the spleen showed slight contamination. Three guinea-pigs were inoculated with emulsions from (a) the site of infection, (b) the regional glands and (c) the internal organs (lung, liver, spleen and kidney). The last mentioned animal (c) died after 2 weeks, yielding no evidence of plague. The other two survived for a month and then succumbed quickly when reinfected with virulent plague cultures.

In spite of these negative cultural and experimental results we may take it for granted that Tb. 29 had contracted plague which it had so successfully resisted that it might almost be said to have reached the point of recovery. Since it is more than probable that the lesions found in the liver were mainly caused by a parasitic invasion of the organ, we may claim that—though the animal had undoubtedly passed through a kind of bacteremic stage—marked changes due to the plague infection were present at the site of infection only. In other words, even when making due allowance for the considerably longer time this animal survived as compared with Tb. 11, we may safely

say that a fundamental difference lies between the two. Tb. 11 displayed the residues of "nodose" plague, while in the case of Tb. 29 evidently no such nodes had formed. Thus Tb. 29 of this series may be classed in the same category as Tb. 5A² of the 1926-7 series, though it is undoubtedly in a more advanced stage. Of great interest is the undeniable resemblance between these two animals (particularly Tb. 29) and those tarabagans developing features of *latent* plague. This point will be discussed later on. For the present, when we try to supplement the experiences made in regard to residual plague of 1926-27 with those observed in 1927-28, we may not only emphasize the existence of this type in the tarabagan but also conclude that recovery from plague infection may be brought about in quite different ways.

E. TARABAGANS SHOWING LATENT PLAGUE

Of the 9 animals now to be discussed seven were killed at varying intervals after infection. In five of these the existence of a peculiar, *latent* form of plague could be proved by experiment. In the remaining two such proof is missing; nevertheless there is strong reason to assume that they also developed *latent* plague. The same holds true of Tbs. 17 and 24 which succumbed spontaneously, their deaths being probably due to accidental causes.

The following table gives some details of the tarabagans under discussion:

TABLE II.

Tb. No.	Died days	Killed days	Results in gps. infected with material fr.		
			Inocd. Site	lymphglands. Regional	Internal organs.
17	12	—	—	—	—
24	14	—	—	Survived.	Survived.
16	—	16	Plague pos.	—	Dto.
25	—	18	—	Survived.	Dto.
26	—	27	Survived.	Plague pos.	Dto.
15	—	28	Plague pos.	—	Dto.
30	—	28	Dto.	Survived.	Dto.
18	—	33	—	Plague pos.	Dto.
19	—	40	—	—	Dto.

² It will be remembered that this animal died accidentally 48 days after infection.

N.B.—Cultures from the internal organs were invariably negative. Rarely, positive or suspicious results were obtained in smears from the site of the inoculation, the bubo or the internal organs.

A detailed description is now appended:

1. *Local reaction.*

Autopsy revealed in every case more or less marked changes at the site of infection. Generally speaking these were more prominent in the animals killed or having succumbed at an early date than in the later ones. In the first group usually some evidence of suppuration was macroscopically evident, while in the second group as a rule some thickening of the skin and congestion or similar alterations of the subcutaneous tissue were observed.

Histologically, the presence of abscesses in varying sizes could be satisfactorily proved in all cases with exception of Tb. 19. In this animal it was only after prolonged search that some minute collections of leucocytes could be found between the superficial layers of the muscles.

Reaction was invariably present round the abscesses, consisting of a slight diffuse infiltration of leucocytes often accompanied by congestion and hemorrhage, occasionally by an increase of the connective tissue. In addition, intracutaneously situated abscesses were detected in animals No. 26, 30, and 18; possibly—though full proof is wanting—a similar process was present in Tb. 19. In the first three cases the leucocytes did not stain well and were embedded in diffusedly-stained masses; evidently these skin pustules were of long standing. It may seem strange at first glance that these pustules were found only in the animals killed long after infection. We must remember, however, that in the others conspicuous local changes were a rule at autopsy, thus leading perhaps to insufficient attention being paid to any slight alteration betraying such skin abscesses to the naked eye.

Suspicious plague-like bacilli were detected in every case. Even in animals, where positive experimental results were obtained with material from the site of infection, bacilli were scarce in the abscesses and still scarcer around them. In Tb. 18 fairly numerous gram-positive cocci—similar to staphylococci—were present in the skin abscess. These cocci in addition to gram-negative bacilli were met with in the deeper layers. In animals 30 and 19 a few gram-positive cocci were found side by side with gram-negative bacilli in the skin abscesses, or in the superficial layers of the subcutis, while only scanty plague-like bacilli were noted in the deeper layers. Probably the invasion of the staphylococci had occurred secondarily in both cases. We

are inclined to think that a similar evolution took place in Tb. 18 as well, as proved by the far greater number of the cocci in the skin abscess than in the deeper layers. It must be noted also that in this case large nests of staphylococci were seen on the surface of the skin. In Tb. 26 only plague-like bacilli were found distributed in small numbers.

2. *Regional lymphatic glands.*

Enlargement of one or a few of the regional glands was seen in some cases, being on the whole more conspicuous in the tarabagans succumbing or killed early than in the later ones. The same seems to hold true of slight peradenitic changes (slight congestion or hemorrhage).

Microscopically few changes were found within the glands except the regular presence of some brownish pigment deposits. Some congestion was noted in Tb. 17, while animals 15 and 16 showed slight hemorrhage. The capsule of the glands was more or less thickened at places with some cell proliferation, occasionally some round-cell infiltration. Slight peradenitic changes were confirmed not only in most of the animals dissected early, but were present also in some of the later ones.

Plague-like bacilli, repeatedly proved to be gram-negative, were found in every instance. Even in the cases where positive results were obtained with material from the glands, the bacilli were few in number, being often detected after a prolonged search only. In cases 18 and 19 a few gram-positive cocci (evidently staphylococci) were found in addition.

3. *Other external lymphatic glands.*

Whenever any uncertainty regarding the other lymphatic glands arose at an autopsy, material from these was preserved for further study.

A matter deserving attention is that positive findings were only obtained in such tarabagans where a longer interval had elapsed between infection and death. The lesions found were analogous to, though as a rule lesser in degree, than those noted in the regional glands. Some suspicious bacilli were present in a majority of the cases, occurring in very small numbers and not always typical in appearance.

4. *Liver.*

The liver of the tarabagans under discussion, while never markedly enlarged, appeared always more or less congested. Whitish capsular thickenings—usually in the form of small specks—were not rarely seen at *post mortem*; sometimes small nodes also. In Tb. 16 one of these appeared calcified: a cyst with size of a pea, filled with a clear liquid and evidently of parasitic origin, was also present.

Congestion of a marked degree was confirmed by histological investigation; sometimes this was most conspicuous beneath the capsule. The latter showed—both in the cases where this had been noted at autopsy and in a few others—spotwise or more diffuse thickenings; cellular proliferation was not marked at such places. A peculiar feature in Tb. 26 was the presence of two small cysts filled with homogeneous, fibrin-like masses, which were situated near one another immediately below the capsule; the latter showed diffuse thickening over this area, while some agglomerations of leucocytes could be noted in the liver parenchyma round the cysts as well as immediately beneath the capsule near them.

Hemorrhages were frequently found. Often they were situated at the periphery of the leucocytic agglomerations or they were in their turn surrounded by a ring of round cells.

Smaller leucocytic agglomerations were found in every case; they were either round and dense, or diffuse and loose. Except in Tbs. 24, 25 and 30, larger leucocytic nodes were present in addition. Some were similar to the small round agglomerations, consisting of closely packed round cells. In everyone of the six tarabagans, either one or a few large agglomerations of leucocytes were seen to surround *parasites* of a peculiar structure. These parasites were evidently identical with those already referred to in Tbs. 13 and 14. They did not resemble the flukes seen in the livers of other species of animals but were apparently round-worms of a not inconsiderable size, the diameter of the transversal sections being 26-32 microns, while the longitudinal sections encountered—though obviously corresponding only to a smaller or larger part of the curled-up parasites, reached a length of 130 microns or more. In the many specimens at our disposal, the parasites were sometimes well preserved, sometimes only broken up fragments were encountered.

In Tb. 24 a whole calcified parasite surrounded by thick layers of connective tissue seemed present. It is not possible to state whether this was identical to the above-mentioned parasites or not.³

Besides the above described nodes containing parasites, others containing none were seen in the six tarabagans. In these leucocytes were less densely massed together, sometimes intermixed with red blood corpuscles. Both red and white cells were embedded in an uniformly stained necrotic mass while round these areas dense agglomerations of round cells were present in which thin layers of connective tissue could be distinguished. Such nodes were not rarely encountered in the immediate vicinity of the parasitic ones.

³ We do not propose to enter here into a detailed description of the structure of the parasites, as this will be dealt with in a special paper.

Eosinophil leucocytes were sometimes noted in the larger nodes. Even on these occasions they seemed practically absent from the smaller agglomerations and infiltrations.

In all nine animals of the series a few suspicious bacilli were found in and near the leucocytic agglomerations of the liver. They seemed as frequent in the large nodes with necrotic changes as in and round the small ones; in some—though not all—instances such bacilli could also be detected in the nodes round the parasites. The suspicious bacilli were repeatedly proved to be gram-negative and seemed usually present in pure culture. An exception was Tb. 18 where a few small groups of staphylococci were noted in addition to the plague-like bacilli.

It can thus be seen that—as compared with the regional lymphatic glands—marked lesions were found in the livers of many of the tarabagans with *latent* plague. It would be too far-fetched, however, to ascribe them all to plague infection. In fact it seems that only the congestion and the small leucocytic formations, commonly present both in the animals apparently free from parasites and these harbouring them, may be ascribed to the plague infection. Such formations are often seen in animals suffering from acute plague. The larger leucocytic nodes, including those with evidence of necrosis, seem principally due to the parasitic invasion. This assumption is not disproved by the fact that positive bacteriological findings were obtained in such nodes. For it is naturally as easy for the *Bacillus pestis* to invade already damaged tissues as to attack normal areas.

5. *The spleen.*

The spleen was usually enlarged and slightly softer than normal; only in a few cases was the swelling marked. Hemorrhage was rarely seen. The follicles appeared often smaller than normal, while the trabeculae were prominent. In a few cases small necrotic areas seemed present. Bacteriologically some suspicious bacilli were found in every case; often they were so scarce as to be detected only after a prolonged search. In Tb. 18 a few gram-positive cocci (arranged in pairs) were noted as well.

6. *The lungs.*

Tbs. 17 & 24 which died spontaneously, deserve special notice. In these two animals areas of congestion could be noted in the lungs at *post mortem*. Histologically bronchopneumonic foci with a partly fibrinous, partly hemorrhagic exudate were noted. A few gram-positive diplococci were seen in and around the pneumonic foci as well as in the peribronchial glands, in addition to some gram-negative plague-like bacilli. As in the case of Tb. 21 there seems little doubt that the death of animals No. 17 & 24 was due to a rapidly evolving lung process of an unspecific nature. In every other respect the two last mentioned

animals displayed the same features as the killed ones.

The latter animals did not show signs of marked pneumonia. Some changes attributable to recent aspiration of blood from forceful killing were often present in the lungs. The congestion, more or less marked in every case, might also be due partly to the forceful death. Sometimes, under the microscope, alveoli singly or in small groups, appeared to be filled, not with fresh blood, but by an exudate partly fibrinous, partly cellular. In the latter case both erythrocytes and white blood cells were present.

In every instance a few gram-negative plague-like bacteria were found in the alveolar walls and in the peribronchial glands. In some gram-positive diplococci—often like *Diplococcus lanceolatus*—were noted as well.

7. The kidneys.

No marked changes were noted at autopsy.

Histologically congestion was always present. Parenchymatous degeneration was occasionally marked. Small areas with round cell infiltration were in a majority of the cases detected in the cortex. Usually, in the same animals small areas were noted in the medulla where formation of connective tissue was seen in addition to congestion, slight leucocytic infiltration and occasional hemorrhage.

A few suspicious gram-negative bacilli were invariably noted in the capillaries especially of the glomeruli. Such bacteria were almost always found in the foci of the medulla and cortex.

It can thus be seen that quite marked alterations were present in the kidneys. [Emphasis may be placed upon the foci in the medulla which as regards position and size correspond to the hemorrhages noted in animals succumbing to acute plague. One is tempted to believe that these lesions present in the animals with *latent* plague were originally of an analogous character and have undergone changes of a reparatory nature.

F. DISCUSSION OF LATENT PLAGUE

Surveying these experiments we may contemplate the alterations found (I) in the internal organs and (II) at the site of infection and in the regional lymph glands.

(I). With regard to the first we are confronted by three facts:

1. That certain lesions due probably to the plague infection were found in such organs as the liver, the spleen and the kidneys.
2. That not only in these, but in all other organs examined suspicious bacilli were found.
3. That on the other hand cultural and experimental tests yielded uniformly negative results.

On account of these negative findings (3), we must ask ourselves whether we have the right to consider the few bacteria found in the organs as plague bacilli. That we really have to do with true *B. pestis* seems proved to us for the following reasons:

1. The bacteria in question, though not always quite typical, were nevertheless similar to *B. pestis* or its involution forms both in morphological and staining properties.
2. It is difficult to see what other bacilli could be so regularly present not only in organs within easy reach of the outside (e.g. lungs), of the intestine (e.g. liver), etc., but also in others more difficult of access (e.g. spleen, lymph glands). A postmortal invasion is out of question in the killed animals, since in their case the internal organs were immediately removed and preserved in formalin. There is at present also no reason to suspect that the organs of hibernating tarabagans are invaded by unspecific bacilli. Similar looking, avirulent bacilli were found in Tb. 29, which awoke a few weeks before it was killed.

We believe therefore that the bacilli found in the organs of the tarabagans with *latent* plague ought to be considered as true plague bacilli. Their presence is in our opinion far less remarkable than the fact that they were invariably avirulent, while on the other hand virulent bacilli were present at the same time at the site of inoculation or in the regional lymph glands.

(II). Turning now to the areas in and near the site of infection it may be well to tabulate certain findings connected with them:

TABLE III.

Results in guinea-pigs infected with material from:

Tb. No.	Inocd. Site	Regional lymph glands.
17	—	—
24	—	Survived (a)
16	Died after 7 days of plague.	—
25	—	Survived (a)
26	Survived (a)	Died after 9 days of plague.
15	Died after 6 days of plague.	—
30	Died after 5 days of plague.	Survived (b)
18	—	Died after 10 days of plague.
19	—	—

Remarks: (a) Was afterwards found susceptible to plague infection.
 (b) Died eventually of accidental causes, showing no evidence of chronic plague.

Before entering into a discussion of these results it is necessary to dwell shortly upon our experimental technique. The material required for the inoculations was kept until the end of the autopsy in sterile, covered Petri dishes, separate dishes being provided for internal organs, regional glands and inoculated site respectively. After the *post mortem* emulsions were made with the aid of previously sterilised normal saline and injected subcutaneously⁴ into guinea-pigs by syringes which had been sterilised before the autopsy. Our invariable rule was to prepare and inject first the emulsion from the internal organs, then from the regional glands and finally from the site of infection.

As can be seen from Tables II & III we could rarely follow our general plan to infect in each case three guinea-pigs with emulsions from the internal organs, the regional glands and the inoculated site respectively. The lesions found at the site of infection and in the glands were often too insignificant or limited in size to yield sufficient material for both histological examinations and animal experiments. Hence the latter had to be sacrificed for the former.

Some conclusions may be drawn from our experiments:

1. From Tables II & III it is gathered that in one case (Tb. 17) no experiments were undertaken at all⁵ while in three (Tbs. 19, 24 & 25) their results were negative. In none of these last three animals was any material from the inoculated site used. On account of this and of the histological findings in all four animals, we feel convinced that they had *latent* plague in the same way as those animals yielding positive experimental results.
2. It is evident that virulent plague bacilli may be preserved not only at the site of inoculation (Tbs. 15, 16 & 30) but also in the regional lymphatic glands (Tbs. 18 & 26). As far as our limited number of experiments enable us to judge, the bacilli may be absent in the latter areas while present in the former (Tb. 30) and *vice versa* (Tb. 26).
3. The virulence of the bacteria preserved as above was undoubtedly rather impaired, the test animals succumbing as a rule considerably later than is usual

⁴ In the experience of Gaiski made during a similar study upon susliks the usual percutaneous method of infection often failed. Gaiski strongly recommends peritoneal injection. This method, though undoubtedly sensitive, leads not infrequently to accidental or premature death of the test animals. We thought it best therefore to compromise by choosing subcutaneous infection.

⁵ We erroneously assumed that the animal dying 12 days after infection had manifest plague and had consequently made no preparation for inoculations.

with subcutaneous methods (the rule being 3 to a maximum of 5 days with our strains). This seems especially true in the cases where gland material was positive.

It will be remembered that the bacilli found in one of the two tarabagans succumbing to plague in 1926-7 after awakening at the normal end of hibernation, showed little if any loss in virulence, while in the other animal they may almost be said to have increased in virulence. If—as we have reason to think—these two tarabagans had *latent* plague while hibernating—we must assume that the plague bacilli preserved during winter at and near the site of infection increased in virulence as well as in numbers in spring and became thus able to invade the internal organs where formerly only few and avirulent bacilli seemed to have been present. Such a possibility is of great theoretical and practical interest.

4. For the reason that the infecting agent was not always strictly confined to the site of infection but may be present in the regional lymph glands we thought it advisable to apply for such cases the term *latent plague* as distinguished from a strictly local affection.

There is much resemblance between the findings made in Tb. 29 (which we believe to have suffered from residual plague) and the tarabagans displaying features of *latent* plague. In fact we consider that no sharp border line exists between the two types. In some of the animals with *latent* plague the bacilli surviving at the inoculated site or the regional glands may lose their virulence, reparatory changes may take place resulting first in *resolving* and then in *resolved* plague. We feel sure, however, that this holds true for only some of the tarabagans thus infected, while in the others the bacilli remain virulent at the site of infection or in the regional glands leading finally to generalised plague at the end of the hibernation period. Perhaps it might have been desirable to observe such animals for longer periods than was possible in the abnormally mild winter of 1927-28. On the other hand, we may claim that the findings established in 1926-7 in the two tarabagans succumbing after awakening coincide nicely with the above scheme and thus firmly support our belief in the existence of a *latent* form of plague among tarabagans.

G. SUMMARY AND CONCLUSIONS

Briefly we may summarise our 1927-8 researches as follows:

1. As in 1926-7 so in 1927-28 (with a specially mild winter) a considerable number of the infected hibernating tarabagans succumbed quickly to acute plague.

Probably this was mainly due to the artificial conditions under which the animals were kept.

2. Experiences gathered from one animal dying on the third day after infection suggest that plague bacilli may invade the general system of the tarabagan quite early and in a gradual manner.
3. Some of the animals showed signs of recovery from the infection. These cases are probably more frequent in nature than under laboratory conditions.
4. A peculiar form of *latent* plague, in which virulent bacilli survive either at the site of inoculation or in the regional lymph glands or both appears to be the principal means of carrying over the disease from one season to another. This may explain the mystery of the perpetuation of plague among hibernating rodents.

THE 1919 CHOLERA EPIDEMIC IN CHINA

BY WU LIEN-TEH

AND

J. W. H. CHUN

A. Historical.

In the "*Nei Ching*" (內經), a famous book attributed to the Yellow Emperor of China (黃帝) who lived about 2600 B.C., five main "disturbances" of the human body were described; namely, of the extremities, heart, lungs, head, and alimentary tract. To the last affection the two characters, 霍亂 (*huo-luan*), were given, and have been handed down to the present day. The real meaning of *huo-luan* is sudden disturbance, which even in those early days, 3,500 years ago, was defined as "a disturbance of the bowels and stomach" (亂於腸胃則爲霍亂).

In a later medical book, published during the Manchu dynasty, called the "Classification of Epidemic Diseases" (溫病條辯), the symptoms of cholera are described as follows: A summer disease, with sudden onset, producing great thirst unrelieved by water, accompanied by repeated vomiting and purging, much sweating, some fever, shivering with cold extremities, contraction of hands and legs, severe cramps, pain over whole body, especially in the abdomen, weak pulse, and delirium. The above seems to fit in well with our modern classical description of cholera!

The Western word "cholera" is evidently a Greek one meaning "spout" which refers to the violent purging like water ejected from the spout of a kettle. The disease was mentioned by Sanskrit writers as far back as 400 B.C., and was also dealt with by Hippocrates. Over sixty references to cholera were also made between 1503 and 1817, but it was not until the latter year that the first authentic account was published of a terrible epidemic, which, first arising in Lower Bengal (the home of cholera, according to Rogers) swept over the whole of India, lasted for five years, and claimed some million inhabitants. From that date onwards, there has been a succession of epidemics of cholera in India, ever extending its boundaries, now to Europe, now to China and Japan, later to America, and lastly Russia, where the disease has established a firm hold.

So far as China is concerned, it may be said that the last great epidemic took place in 1902, as part of the pandemic which

passed over Arabia, Egypt, Syria, Persia, Singapore, the East Indies, the Philippines, Japan, and Formosa. In 1908, another great pandemic overtook Russia and many parts of Europe, but only a few cases were registered in Shanghai.

Cholera being essentially a water-borne disease, this disease does not play such havoc among the Chinese (who are tea-drinking people) as among inhabitants of Russia, India, Arabia, and similar countries where the drinking of unboiled water is the rule. It may therefore be understood that although outbreaks of cholera are quite common in Japan and the Philippines, China, except under exceptional circumstances, is fairly free.

The great epidemic of last summer, which swept over the whole country and reached Japan, Siam, India, Afghanistan, Russia, Sweden, etc., may be said to have been introduced from the Philippines to Swatow and Foochow. In the latter city, the first cases were reported early in July, the number rapidly increasing, until nearly a thousand cases were supposed to have occurred daily during the height of the epidemic. From there, it traveled northward and southward. So far as our records go the following are approximately the dates when the first cases appeared in each city:

Foochow	about July	7
Shanghai	„	15
Yinkow (Newchwang)	„	22
Mukden	„	24
Dairen	August	3
Harbin	„	5
Changchun	„	15
Kirin	„	16
Langfang	„	15
Fengtai	„	15
Tientsin	„	20
Peking	„	20
Sansing	„	22
Tokyo, Osaka, Moji	„	28
Seoul (Korea)	„	29

Outbreaks were reported from many inland cities in Honan, Anhwei, Hupeh, and Hunan in a more or less virulent form. Altogether the number of deaths due to cholera during the summer months could not be less than 300,000, equal perhaps to the toll from influenza last year.

Our notes on the cholera outbreak have been based upon our experiences in Harbin, but as the principal features are some-

what similar in other districts affected they may be taken as fairly representative of the disease as a whole.

The port of entry of the disease last summer, so far as Manchuria is concerned, may be said to have been Newchwang, which reported its first case on July 22. From the beginning it showed the greatest virulence and claimed a large number of victims. Persons incubating the disease or actually suffering the disease escaped to other places, principally by rail, and spread the infection broadcast. As the weather this year was one of the hottest and driest on record, the people consumed all sorts of fruit, particularly melons, and drank unboiled water carelessly. Moreover, the number of flies was unusually great and proved a most important factor in the dissemination of the infection. For it is well known that in the north of China, and especially the newly opened parts of Manchuria, the poorer classes had no proper places reserved for their evacuations. To them, any open spot is good enough and the germs of cholera are thus conveyed broadcast by the flies feeding on feces. Melons, especially muskmelons, were displayed on the ground covered with contaminated earth, and eaten, skin and all, before they were even washed. Owing to the dryness of the weather, the wells were drier than usual and the water was therefore more easily contaminated. No wonder cholera spread like wildfire and claimed in Harbin alone over 4,500 deaths in just over six weeks.

The part played by the flies was probably a mechanical one, the germs sticking to their legs when they fed on infected stools and being later released on exposed food.

B. Symptomatology.

The clinical features of cholera are perhaps familiar to most. The following represents a typical case: After an incubation period lasting from two to three days the patient complains of being unwell, may have slight fever, and suffers from slight diarrhœa and pain in the stomach. This may go on for a few hours or even a day or two. Suddenly, he experiences great pain in the belly, and severe diarrhœa and vomiting follow. The stools may be yellowish at the beginning, but soon become very liquid and assume a lighter and looser character, being sometimes ejected like water from the tap. They may even be blood-stained. The vomiting is continuous, the ejected material consisting at first of food taken, but later of a simple watery fluid with or without bile. The patient complains of great thirst and rapidly becomes exhausted. Severe cramps of the back, leg, and other muscles occur. His features become shrunken, the skin assumes an ashy-gray hue, the eyes are sunk in the orbits, the nose is pinched, the cheeks are hollow, the voice becomes husky, the extremities turn blue, and the skin is shriveled and covered with

a clammy perspiration. The pulse may not be felt, and no urine may be passed at all. The patient may die from auto-intoxication and heart failure, or may pass on to a comatose stage from which he does not recover.

If medical aid is given in time or if the patient shows signs of recovering, a reaction sets in. The blue color gradually disappears from the face, the skin becomes warmer and pinker. The pulse becomes stronger, and both diarrhœa and vomiting gradually disappear. For some time afterwards the patient remains in a very weak state, requiring careful nursing.

Our studies of the epidemic in Harbin brought to light some interesting features, which may be briefly described here:

C. Bacteriology.

When the epidemic at first broke out we thought it was something like the *Choleraic Diarrhœa* which had been raging in Shanghai, and in which the characteristic vibrio could not be isolated. During a recent visit to Shanghai, one of us learned from Dr. Tyau, in charge of the Pathological Department of St. Luke's Hospital, that he had only succeeded in isolating the cholera bacillus from one case, and even that one culture refused to grow for a second generation.

In Harbin, however, we had no difficulty in isolating the bacillus, for on ordinary agar plates it could be easily distinguished as bluish-gray colonies and transplanted. In some cases, a pure culture could be obtained straight from the stools without previous plating. Altogether, we examined twenty-one stools, and isolated the cholera bacillus in nineteen, the other two being both cases of bacillary dysentery from which the causative organisms were isolated. The cholera cultures obtained by us were subjected to all necessary tests, such as agglutination (positive up to 1 in 12,000), animal, cholera-red, gelatin, etc., and all responded. The majority of organisms obtained by us seemed to be rather more slender than what is commonly known as the typical bacillus, but their curved appearance, morphological characteristics and their staining and other reactions were unmistakable.

D. Pathology.

This will be treated in a later article.

E. Clinical Features.

Out of the 13,000 odd cases occurring in Harbin, we admitted into our Plague Prevention Hospital 1,822 males and 149 females.

Of these the majority in both males and females were between twenty-one and forty years of age. See table.

Of the deaths, those between twenty-one and forty years also numbered most.

There was relatively a smaller number of deaths among females than among males.

A fine physique was apparently no protection against the disease; in fact, such patients seemed to withstand the effects of the toxin more poorly than thin persons. Some of our worst cases were among those who weighed 180 pounds or more, and these developed uræmia or died suddenly of heart failure. They also seemed to suffer more from the after effects of saline infusion. An interesting exception was the case of a tall Customs boatman, who got cured after four critical days. See illustrative cases.

The epidemic claimed victims from all classes of people. The only way of catching it was to eat infected food or drink infected water. Some of the prominent merchants died, and two of the members of the Anti-Cholera Committee were missing on the second day of the meeting because they had died of the disease in the interval. The former Taoyin of Taheiho nearly died.

Three ladies, who ate raw shrimps *à la Shanghai*, were all attacked by cholera, and one died.

The epidemic showed a marked decline after the onset of rain. Its worst period coincided with the largest number of flies. After the rain, the flies also diminished in number.

The stools of cholera have been compared to rice water. This is not strictly correct, as the stools, though white and watery, are clear and transparent, not opaque and cloudy as rice water. A good description of a cholera stool would be one which is almost transparent, or slightly opalescent when seen in a glass vessel, having little or no odor, but a faint meaty smell, and containing white flakes, consisting of mucus, which settle down when left alone. The patient may pass from ten to fifty or more stools in the course of twenty-four hours, leaving him prostrate and absolutely dried up.

The vomit may at first consist of the usual contents of the stomach, then bile-colored watery or sticky fluid, and later simple sticky fluid quite pale in color.

Microscopically, the opaque stools are seen to be due to the presence of fine granules derived from broken-down epithelial cells. The sediment consists principally of mucus and these damaged cells in various stages of granular degeneration.

The *vomiting* is one of the most distressing features of cholera, and the constant and voluminous ejections of a watery fluid, when none has been swallowed, is one of the most pathognomic features of the disease. The repeated call for water and its equally quick rejection add to the strain. It is found that frequent sips of water are more easily retained, and that small doses of tincture of iodine relieve the symptom.

Abdominal pain, often in the epigastric region, is another prominent symptom, and may be due to the cramps of the abdominal muscles or the abdominal contents or both.

The *muscular cramps*, another pathognomic feature, may be felt from the beginning and continue to the last stages of collapse. They are confined principally to the muscles of the abdomen and extremities and are seldom felt in the face or back. Well-developed persons feel these cramps more, which may be considerably relieved after saline infusions.

The surface *temperature* is much reduced, although *per rectum* the thermometer may mark 99° F. or more. The pulse quickly loses its steadiness and in bad cases is not felt at all.

The *blood pressure* is often below 70 mm. and sometimes not registrable.

Hiccup is troublesome, but not necessarily a bad omen. In two cases of strongly built men, ending fatally, we observed marked bellowing, like the lion's roar, for ten hours before they died. Both appeared unconscious of their surroundings.

Suppression of the urine follows the fall in blood pressure and is a serious feature of the disease. If unrelieved, this soon ends in uraemia and finally death of the individual. The toxin of cholera apparently has a disastrous effect on the kidneys, which condition is aggravated by the continuous evacuations, vomiting, and consequent increased density of the blood.

Albumin was found in at least half of the cases examined (44); some in small, others in large quantities. Not all the albumin cases died, however, and at least half of the cases showing no albumin in the urine died. See table.

Owing to the desire of a large number of patients to return home within twelve to twenty-four hours after injection it was somewhat difficult for us to observe accurately the stages of collapse and reaction among them, but among those who remained in the hospital we found that the longer the collapse stage lasted the greater was the danger of complications arising, especially uraemia. To these we repeated the infusions and also performed proctoclysis (injection of salines by rectum) wherever possible.

We also noted several cases of typhoid-febrile reactions.

The voice gradually loses its strength and may be lost entirely.

F. Diagnosis.

The bedside symptoms of cholera are sudden vomiting and violent purging of white liquid mucous stools, muscular cramps, pain of abdomen, loss of voice, and complete collapse of patient. In a well-established epidemic there ought to be no difficulty in diagnosing the disease, but in case of doubt, especially at the beginning of an epidemic, the bacteriological diagnosis should always be made. Microscopical examination of the stools is not sufficient; plating should always be made and suspected colonies subcultured. Cholera colonies are semi-transparent, pale, bluish-gray in color. Stained films of the organisms show the characteristic comma-shaped bacilli, while those grown in peptone water display marked motility, in hanging-drop preparations.

During the last epidemic in Harbin, we encountered a large number of cases simulating cholera, such as the following:

(a) *Dysentery*. Both amœbic and bacillary. One Japanese boy of ten years old was admitted with diarrhœa, vomiting, and collapse. Cultivations from his stools showed only *Dysentery bacilli*. Infusions of saline were also given besides the usual drugs for dysentery, but he died six days after admission.

(b) *Typhoid*. Of which we had four cases at the beginning of the epidemic. Later on, when cholera was on the wane, the number of typhoid cases increased, as the hot season and large number of flies seemed to favor the propagation of typhoid and dysentery as well as cholera.

(c) *Simple Enteritis*. Several cases of simple enteritis were seen at the hospital, but these returned to their homes after being assured that their disease was not the dreaded cholera.

(d) *Tubercular Enteritis*. Two cases of this disease were seen, one a man of thirty-one with marked pulmonary complications.

(e) *Morphinism*. We encountered over ten cases of morphine *habitués*, who during the epidemic, found themselves deprived of their drug and their hiding places. Profuse diarrhœa set in, and some gave a history (perhaps false) of abdominal pain and cramps. In three cases, actual cholera symptoms were noticed.

(f) *Typhus*. Six cases of typhus were admitted during the epidemic with no deaths.

(g) *Pneumonia*. One case of marked lobular pneumonia in a fat, well-built shopkeeper was admitted, because there was no other place for the poor man to go. He died twenty-four hours after admission.

It may seem strange that such varied cases as the above were admitted into a cholera hospital during an epidemic. Harbin is, however, full of crowded inns and shops, and during an epidemic, when hundreds of persons were taken ill daily, the inmates, suffering from fever, abdominal pain, diarrhœa, or any ailment like cholera, were turned out, and the only place they could find admittance was our hospital. It was fortunate that at no time, even during our busiest times, did we have to refuse admission to urgent cases of any character.

G. Treatment.

Details of treatment as adopted by us during the epidemic, as well as the general management, will be treated in a separate article, but we may mention here that besides giving solutions of permanganate of potassium by mouth to oxidize the contents of the stomach and iodine tincture to allay the vomiting, we employed Rogers' method of saline infusion on an extensive scale. This solution is made up as follows:

Sodium Chloride	120 grains	(8 grams)	
Calcium	„ 4	„ (0.25 grams)	The results were
Potassium	„ 6	„ (0.40 „)	very favorable.
Distilled water	1 pint	(568 c.c.)	

Two to three litres of this solution were infused into the brachial veins through a salvarsan needle by the siphon method (two glass tubes fixed through a rubber cork into a large five-pound bottle) or even by means of a large-caliber needle attached to the rubber tubing of a glass enema apparatus. This infusion was repeated whenever necessary; in some cases we repeated the operation four times before the patient was finally relieved.

We did not use the Kaolin treatment as that drug was not procurable at the time.

Tables of Statistics Appended to Cholera Article

1. Meteorological Readings of August, 1919.
2. Chart Showing Temperature and Mortality Incidence.
3. Age and Sex Incidence of Admissions.
4. Age Incidence of Mortality.
5. Albumin Incidence as Shown in Urine Examinations.
6. Hospital Admission and Mortality in Various Hospitals.
7. Relative Number of Deaths Among Population.
8. Estimated Population in Harbin Area.
9. Statistics of Anti-Plague Hospital.
10. Statistics of New Fuchiatien Hospital.
11. Statistics of Russian Hospitals.

(1) Meteorological Readings

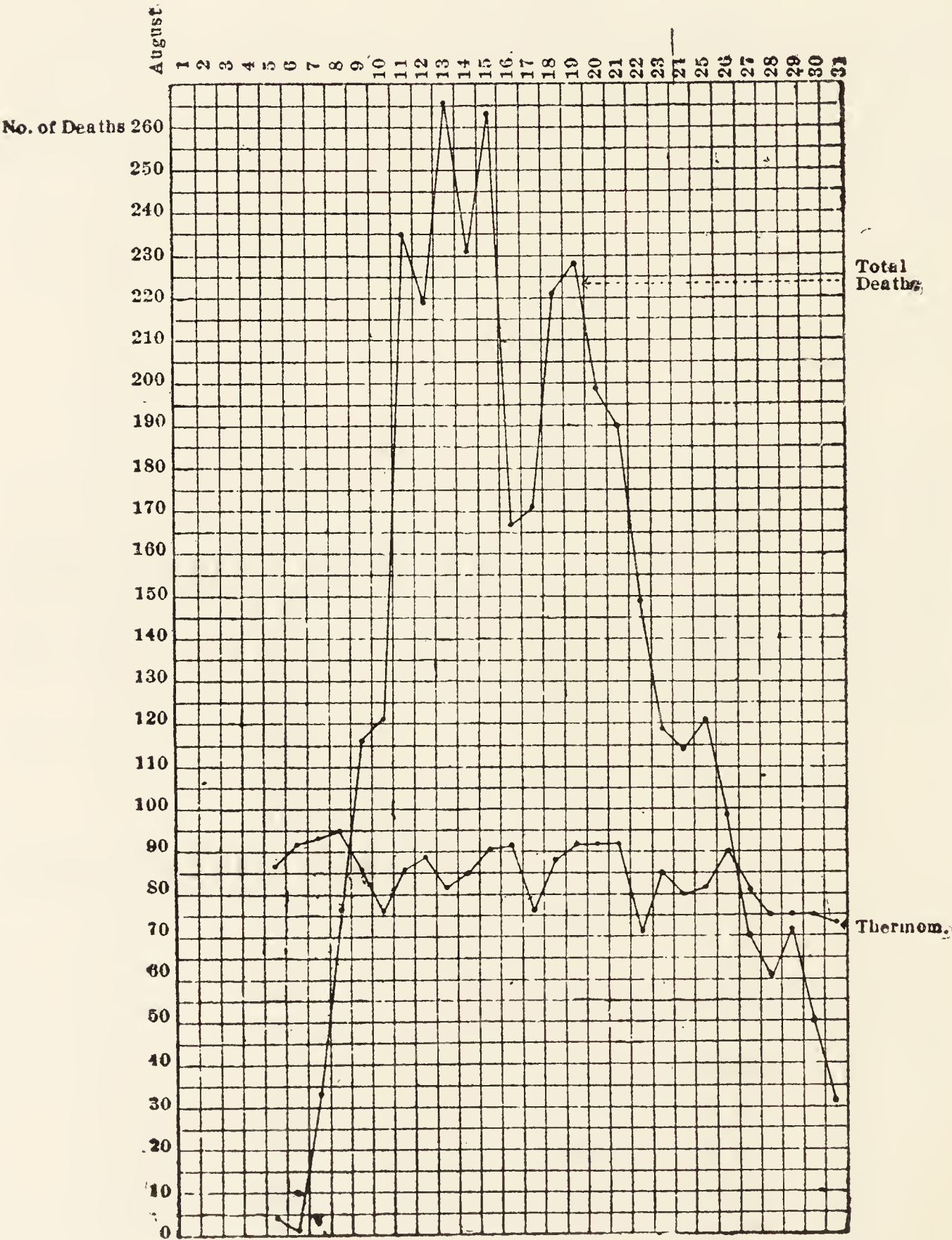
From August 1—31, 1919

		BAROMETER		THERMOMETER	
<i>Date</i>		<i>Highest</i>	<i>Lowest</i>	<i>Highest</i>	<i>Lowest</i>
Aug. 1.		29.552	29.466	85	61
	2.	29.490	29.372	81	67
	3.	29.395	29.296	84	64
	4.	29.353	29.328	88	66
	5.	29.497	29.417	87	63
	6.	29.537	29.431	93	68
	7.	29.501	29.466	94	69
	8.	29.558	29.436	95	73
	9.	29.502	29.464	86	74
	10.	29.534	29.488	77	66
	11.	29.357	29.494	86	64
	12.	29.532	29.464	88	60
	13.	29.601	29.437	83	71
	14.	29.606	29.467	85	61
	15.	29.460	29.353	91	61
	16.	29.350	29.257	92	69
		rainfall for 24 hours without stopping.			
	17.	29.352	29.302	78	58
	18.	29.485	29.362	88	55
	19.	29.550	29.497	93	64
	20.	29.642	29.605	93	65
	21.	29.622	29.522	93	64
	22.	29.538	29.350	73	67
		heavy rain for 36 hours.			
	23.	29.462	29.420	85	65
	24.	29.470	29.390	80	72
	25.	29.522	29.474	82	63
	26.	29.524	29.416	90	65
	27.	29.503	29.432	81	57
	28.	29.496	29.380	75	56
	29.	29.394	29.366	75	54
	30.	29.432	29.263	75	54
	31.	29.455	29.335	74	53

Barometer Readings: Highest 29.642 on the 20th at noon.
 Lowest 29.257 on the 16th at 3 p.m.

Thermometer Readings: Highest 96 on the 8th.
 Lowest 53 on the 31st

(2) Temperature and Mortality Incidence
Harbin Cholera Epidemic, 1919



(3) Admissions. Age Incidence				
	MALES. 1,822		FEMALES. 149	
	No.	%	No.	%
1-10 yrs.	49	2.7	11	7.3
11-20	179	9.8	20	13.4
21-30	592	32.5	39	26.2
31-40	581	31.9	36	24.3
41-50	290	15.9	21	14.2
51-60	116	6.3	18	12.0
61-70	13	0.7	4	2.6
71-80	2	0.02	0	0.0
81-90	0		0	
Total	1,822	100.0	149	100.0

(4) Mortality. Age Incidence				
	CHINESE CITY		ANTI-PLAGUE HOSP	
	No.	%	No.	%
1-10	20	1.3	1	0.4
11-20	134	8.8	9	3.5
21-30	345	22.6	90	34.7
31-40	454	29.8	78	30.1
41-50	333	21.8	63	24.3
51-60	176	11.6	12	4.6
61-70	53	3.5	4	1.5
71-80	8	0.5	2	0.8
81-90	2	0.02	0	0.0
Total	1,525	99.9	259	99.9

(5) Urine Examinations for Albumin

Series I (Before September 1 when epidemic was at its height):

. Cases Exd. No Albumin Trace Albumin Much Albumin Deaths

33 13 6 14 6

Series II (After September 1 when epidemic had much abated):

11 5 5 1 2

Of the deaths recorded above, the urine examination results are:

Series I 6 3 1 2
Series II 2 1 0 1

(6) Hospital Admissions (Cholera Epidemic, 1919)

Hospital	Admissions	Deaths	Percentage Mortality
Russian Central	400	135	33.75
Russian Municipal	180	104	57.77
Chinese Anti-Plague	1,962	275	14.11
New Chinese	185	33	17.89

(7) Relative Number of Deaths in Whole Harbin Area

<i>Nationality</i>	<i>Location</i>	<i>No. of Deaths</i>
Russian	Railway Area	705
Japanese	Railway and Chinese Areas	60
Chinese	Pristan	507
	New Town	115
	Chinese city	2,799
	Anti-plague H.	273
	New Chinese H.	35
	Railway wagons	9 3,738
		<hr/> Totals 4,503

(8) Estimated Population of Harbin Area
(Chinese and Russian cities)

Russians	35,000
Chinese	95,000
Japanese	3,000
Other nationalities	2,000
		<hr/> Total 135,000

(9) Plague Prevention Hospital (*August 5 to September 10*)

<i>Date</i>	<i>Admissions</i>	<i>Deaths</i>	<i>Discharged</i>	<i>Under Treatment</i>
Aug. 5.	1	0	0	1
6.	0	0	0	1
7.	1	0	0	2
8.	2	0	0	4
9.	8	3	5	4
10.	9	2	3	8
11.	29	7	12	18
12.	57	6	26	43
13.	69	4	34	74
14.	92	9	45	112
15.	107	11	81	127
16.	58	9	57	119
17.	117	10	87	139
18.	133	21	90	161
19.	124	28	120	137
20.	123	19	112	129
21.	112	19	105	117
22.	99	17	90	109
23.	137	15	104	127

<i>Date</i>	<i>Admissions</i>	<i>Deaths</i>	<i>Discharged</i>	<i>Under Treatment</i>
Aug. 24.	132	15	98	146
25.	77	22	73	128
26.	85	11	89	113
27.	66	9	84	86
28.	55	10	61	70
29.	65	11	53	71
30.	31	3	54	45
31.	32	2	28	47
Sept. 1.	32	3	46	30
2.	26	1	19	36
3.	19	4	25	26
4.	13	0	13	26
5.	16	1	21	20
6.	9	2	8	19
7.	9	1	13	14
8.	6	1	5	14
9.	6	0	8	12
10.	5	0	1	16
	<hr/> 1,958 <hr/>	<hr/> 275 <hr/>	<hr/> 1,670 <hr/>	<hr/> 2,343 <hr/>

(10) New Fuchiatien Hospital (*August 25 to September 13*)

Aug. 25.	9	0	1	8
26.	8	4	1	11
27.	7	1	4	13
28.	25	1	14	23
29.	23	10	21	15
30.	8	4	6	13
31.	9	2	8	12
Sept. 1.	6	3	1	14
2.	16	2	15	13
3.	13	1	11	14
4.	9	1	12	10
5.	13	1	11	11
6.	5	1	6	9
7.	6	0	6	9
8.	7	0	6	10
9.	5	2	5	8
10.	5	0	8	5
11.	5	0	6	4
12.	6	0	10	0
	<hr/> 185 <hr/>	<hr/> 33 <hr/>	<hr/> 152 <hr/>	<hr/> 202 <hr/>

(11) Cholera Report of Russian Central Hospital

		<i>Dis-</i>			
<i>Date</i>		<i>Admitted</i>	<i>charged</i>	<i>Died</i>	<i>Remaining</i>
1919 Aug.	10.	12	0	1	11
	11.	5	2	1	13
	12.	5	3	2	13
	13.	17	2	10	18
	14.	12	4	4	22
	15.	12	4	4	26
	16.	15	1	5	35
	17.	12	3	7	37
	18.	10	1	7	39
	19.	16	2	2	51
	20.	17	3	4	61
	21.	19	3	18	59
	22.	17	3	2	71
	23.	17	4	2	82
	24.	10	0	0	92
	25.	34	11	3	112
	26.	26	1	7	130
	27.	25	5	7	143
	28.	13	7	2	147
	29.	19	13	7	146
	30.	12	9	7	142
	31.	9	5	1	145
Sept.	1.	13	6	9	143
	2.	6	14	2	133
	3.	5	16	5	117
	4.	10	12	6	109
	5.	3	12	0	100
	6.	7	10	0	97
	7.	11	9	1	98
	8.	1	7	3	89
	9.	2	11	1	79
	10.	2	4	1	76
	11.	1	8	1	68
	12.	1	0	0	69
	13.	1	0	1	69
	14.	2	0	0	71
	15.	1	0	0	72
	16.	0	5	2	65
Total		400	208	135	65

Russian Municipal Hospital

No individual tables are obtainable, but it was acknowledged by the medical authorities that the totals are as follows:

Up to September 10	Admission	180	
	Deaths	104	
	Discharged	62	
	Remaining	14	Mortality % 57.77.

THE CHOLERA EPIDEMIC OF 1926

BY WU LIEN-TEH, J. W. H. CHUN, R. POLLITZER

General Survey

About the middle of May information reached us that a few cases of Asiatic cholera had occurred among residents of the Chapei District in Shanghai, but nothing official was declared until June 8, when the Settlement authorities reported their first authentic case of cholera which had been admitted into hospital four days previously. From that date onwards the infections began to accumulate until the summit was reached about the week August 1-8, during which 2,481 cases were reported as having been treated at the two Chinese Summer Diseases Hospitals. Altogether no less than 20,000 cases have occurred in the Shanghai district alone. Serious outbreaks were reported in almost every city in the Yangtze area, such as Nanking, Soochow, Wusieh, Huchow, Anking, Hankow, Wuchang, etc., as well as farther south in such centers as Foochow, Amoy, Swatow, Canton, Hainan, etc. The country of Siam reported 5,000 in deaths alone.

It may be remembered that a similar outbreak of cholera, affecting several thousand victims, occurred in Shanghai the year previous (1925). I happened to be then in that city in August and secured samples of water from the Chapei Waterworks (Soochow creek, filter beds and tap) and requested the Municipal Health Laboratory to make a bacteriological examination of some for cholera. The report was that although suspicious vibriones were cultivated from every sample they were probably not of the pathogenic variety. In July of this year, when the cholera situation was becoming acute, Dr. Noel Davis, the Health Commissioner, had samples of the Chapei Waterworks bacteriologically examined and reported that in every one (intake from Soochow Creek, effluent from filter beds and a tap from the mains) specific cholera vibriones were isolated. Much acrimonious controversy has since arisen because of that discovery, but it seems to me that the real problem before the health authorities of the International Settlement and the Chinese area is effective coöperation in

- a. An intensive study of suspected sources throughout the year, including water, carriers, etc.
- b. Preventive measures to be undertaken early in the spring.

- c. Mass prophylactic inoculations.
- d. Early notification to non-endemic centers, so as to limit the spread to other localities.
- e. Other problems, e.g., mass fecal examination at ports and railway centers.

No unprejudiced person will deny that the present loose laws regarding notification and quarantine, which were probably devised half a century ago, need urgent revision. For the sake of all concerned, including the various foreign authorities and commercial bodies in China, it is hoped that a cholera prevention conference will soon be held in or near Shanghai so that all cards may be placed on the table and really effective preventive measures may be devised against this almost yearly visitation of an easily controllable disease. Should a proper system be established such incidents as when the British steamer *Lienshing* tried to smuggle a dying cholera patient ashore on August 3 resulting in a fine of \$250 to the captain and twelve months' rigorous imprisonment to both the first and second compradors might not occur.

We may now proceed to a study of the epidemiological and clinical data before us. As has been said above, the first case was officially declared in Shanghai on June 8, though it was clear that isolated cases had occurred since the middle of May. A state of epidemic was not reported until July. In the meantime, ports in Chekiang, Fukien, and Kwangtung, as well as in the Yangtze valley, had been invaded. The first cases apparently did not reach the northern cities until the first week of August, though Tientsin reported its first case on June 15. The following table showing (a) when quarantine was first declared at various northern cities and (b) when the first case occurred in them, respectively, is highly illuminating:

Locality:	Quarantine		Quarantine Stopped
	Declared Against Shanghai	First Cholera Case	
Dairen	July 22	Aug. 28	Oct. 11
Antung	„ 29	Sept. 7	—
Newchwang	„ 19	Aug. 19	Oct. 10
Tsingtau	Aug. 9	„ 6	„ 15
Japan	„ 2	„ 3 (S. S. Macedonia)	—
Tientsin	—	June. 15	—
Harbin	—	Aug. 5	Sept. 15
Port Arthur	July 22	„ 27	„ 24
Changchun	—	„ 14	„ 18

The Japanese authorities at Dairen enforced fecal examination of passengers and crews of steamers arriving from

infected ports, on July 22. Up to September 30, they had examined

259 Steamers with Crews totaling	15,314
548 Junks " " "	3,091
Passengers, Male	18,829
" Female	2,861
<hr/>	
Total	40,095

The South Manchurian Railway authorities also enforced fecal examination of railway passengers arriving from the north beginning August 23; this was stopped on September 18.

The above table shows, among other things

- a. That quarantine measures were in most cases applied much too late.
- b. That even with this delay the cases in each center appeared after a considerable interval.
- c. That cholera outbreaks when they did occur were of comparatively short duration, the end coinciding often with the coming of cold weather.

The whole 1926 cholera epidemic probably claimed only 1,500 victims in Manchuria as compared with over 10,000 in 1919; this is specially marked in the case of Harbin, which had 4,500 in 1919 and only 280 in 1926. The only severely affected centers were Antung (480), Kirin (320), Harbin (280), Changchun (210), and Yinkou, (167). Other cities of Manchuria show a corresponding decrease. This satisfactory state of affairs may be traced to

- a. Early preventive measures by the medical authorities.
- d. Good understanding between the civil and medical authorities.
- c. Hearty coöperation between the Chinese and the Japanese health personnel.
- d. Effective educational propaganda among the masses who willingly receive prophylactic inoculations and send their patients early to hospital for treatment.
- e. Perhaps prevailing wet weather during the second half of the summer.

Turning now to our Harbin experiences, we may class them under three headings, namely:

- A. Epidemiological Study
- B. Cholera Notes from Pinchiang Hospital
- C. Laboratory Reports

The data gathered are not complete but we hope to supplement these in a later article. It is hoped that each section may contribute something to the knowledge of this disease in China, particularly the ease with which the disease may be bacteriologically diagnosed early in an epidemic, and also the

successful result of the hypertonic saline infusion. Lastly, the need for detecting early carriers, especially at sea and river ports, seems to us all-important. For this reason we support the policy of mass fecal examination adopted in Dairen for passengers and crews of steamers arriving from infected localities.

WU LIEN-TEH.

A. Epidemiologic Study of the Cholera Epidemic in Harbin, 1926

(To be read with Tables I-XIV)

Mortality Figures

As can be seen from Tables I and II there is a great difference in the mortality observed in our hospital (17.3 per cent) and that of the patients admitted in the Municipal Hospital (54.5 per cent), while the Railway Hospital takes an intermediate position with 34.5 per cent fatal cases.

A similar discrepancy between our records and those of the Russian hospitals was evident also in the epidemic of 1919 (57.77 per cent and 14.11 per cent). This appeared fully accounted for by the differences of therapy used, our favorable results being due to the use of *hypertonic intravenous* salt infusions, while the Russian doctors at first stuck to *subcutaneous* administration of normal saline. Though, as will be shown later, the question of the therapy employed played probably a rôle also in the 1926 epidemic, this factor alone cannot account for the differences of mortality observed. In this epidemic hypertonic intravenous infusions were also given in the Municipal and Railway hospitals. Specially the doctors of the former took great pains to ascertain our methods of treatment and to adopt them for their patients.

When analyzing the records of the 1926 epidemic we have to consider that possibly a better resistance of Chinese patients against cholera may account for our results. In fact, in the Municipal Hospital, where both foreigners (mainly Russians with a few others like Jews, Poles, Armenians, and one Korean) and Chinese were admitted, the former had a mortality of 57.5 per cent, the latter of 50 per cent. In the Railway Hospital this is still more striking, the figures being 43.5 per cent and 28.1 per cent, respectively (see Table I). Since cholera is a rare visitor of North Manchuria and Shantung (which supplies the majority of our coolies) and a natural immunity cannot be expected, allowance must be made for the existence of a stronger resistance among Chinese. However, in our opinion, this cannot wholly explain the great difference in the figures, for two reasons:

1. The mortality among the Chinese admitted in the two other hospitals is far higher than that in our own (see Table I).
2. If such a resistance should exist to a marked degree, slight cases ought to preponderate among our patients. This, however, was not the case, the majority of our sick presenting typical symptoms of cholera. Also, there is no reason to assume that only the serious cases reached our hospitals, a majority of slight ones remaining unnoticed at their homes.

What other factors could then have been at work? In order to elucidate this question it will be well to analyze step by step the figures available.

a. Sex Incidence.

Table III shows that in the Municipal Hospital more foreign females were admitted than males (65.0 per cent as against 35.0 per cent). Naturally the thought arises that perhaps a greater mortality among the former might account for the high total mortality. Table IV, however, shows just the contrary. Also in our hospital and in the Railway Hospital the mortality of males was perceptibly, though not so markedly, higher than that of females. This table also shows that both in the Municipal and Railway hospitals the mortality of foreign males was considerably higher than that of the Chinese. This might be explained in part by the better resistance of the latter. Other factors are probably at work as well; these will be discussed later on.

b. Age Incidence

While scanning the records of age incidence (see Tables V and VI) we find no differences worth considering. Both among Chinese and foreigners the incidence of the disease and the mortality are highest in the young adult.

c. Occupation

A study of the occupational incidence (unfortunately only limited figures are available) does not teach us much (see Table VII *a* and *b*). The most striking figures in the Russian table are those recording an outbreak among 12 patients who were in the general hospital and two hospital employees. This will be discussed separately, but we may state here that the mortality among the patients was even slightly lower than that of the outside admittances (see Table VIII). The incidence among persons out of work is comparatively high (22.5 per cent—VII *b*). However, here also the mortality is not above the average (about 50 per cent).

d. Time of Admission

A fundamental difference existed in 1926 in regard to the system of admission in Pinchiang on one hand and in the Special Area (Municipal and Railway hospitals) on the other. In the Special Area the patients were mainly brought compulsorily to the hospital and kept there—if necessary by force—as long as deemed necessary (see Table IX). In Pinchiang we did not even attempt to adopt such a rigid scheme. Our policy was to appeal to the population to seek our aid by all available means (personal urging through our sanitary inspectors, distribution of handbills, posters, etc., all over the Chinese town, etc.). That our campaign was successful may be gathered from several facts:

1. Seventy-six per cent of 85 patients for which we have exact data sought admittance during the first two days of illness (see Table X).
2. A comparatively large number of patients came who suffered not from cholera but from other bowel complaints (see Table XI). However, they had heard of our successes and—being afraid to run unnecessary risks—availed themselves of our advice and treatment.
3. Actually we can assert that 90 per cent of the cholera cases in Pinchiang went through our hands. Certainly they benefited by our treatment, which was given free of charge without any hindrance to the personal liberty of themselves or friends.

Our colleagues of the Municipal Hospital complained that the compulsory system led, not to a speedy dispatch of the cases to the hospital, but to the hiding of the same and asserted that the delayed admittance of the patients was to a large degree responsible for their unfavorable results. They assert that besides the cases arising in the hospital itself only very few patients were admitted on the first day of illness. A study of Table X shows, in fact, that the percentage of their first and second days' admission was less than ours. We do not think, however, that the differences are marked enough to account fully for the discrepancies in the mortality figures. Moreover, our figures could be based upon about half of the cases only. Finally, the percentage of mortality according to the day of illness (see Table X, last columns) shows in both hospitals such strange oscillations that one must hesitate to draw any final conclusions.

Summarizing, we may say that the facts so far available do not fully explain the differences in the mortality figures. We proceed now with a discussion of two other factors which are of great importance.

e. Physical Status and Mode of Life of the Patients

Our own patients were mainly of a modest social standing with ample opportunity for exercise through work and in no danger of overfeeding; likewise, alcoholics were very rare among them. Our Russian colleagues assure us that they had a very high percentage of habitual drinkers. During our visits to the Municipal Hospital we could notice such cases as well as specimens of Russians who believe in eating as much as possible, considering floridness as a sign of good health. It is well known that drunkards fall an easy prey to cholera. Likewise, it has been our experience in this as in other infectious diseases that it is not the florid and markedly well nourished, but the wiry individuals who stand a good chance of overcoming infection. These factors may explain in part the high mortality among foreign males, but they cannot be generally accepted for Chinese (see Table IV *d*). Certainly only a part of the latter, if any, adopted the eating and drinking habits of the Russians among whom they lived.

f. The Problem of Treatment

Our organization stands in the first line for the fight against epidemics. Both our staff and our equipment are in constant preparedness for such, so that we can take up the work at short notice. In the Russian Hospital no such ready organization existed; the wards for infectious diseases were filled with patients and had to be evacuated under great difficulties to make room for the influx of cholera patients; a new staff had to be organized to take charge of the latter. These difficulties certainly reflected upon the quickness with which treatment was given, as we saw personally that delays were inevitable for some time until a routine was created.

Our patients, who came voluntarily, reached the hospital quickly without having to go through formalities or having to wait for the ambulance, etc. In the hospital we took them at once to the infusion room, if necessary without insisting upon bath, change of clothes, elaborate examination, etc. Likewise everything was ready day and night for the quick repetition of the infusions. We have reason to assume that the procedure in the Municipal Hospital was a long one.

Cholera Outbreak Among Inmates of the Municipal Hospital*

An unusual and alarming feature of the 1926 epidemic was an outbreak among inmates and employees of the Municipal Hospital (see Tables VIII and XII).

This solidly built hospital is situated in the Special Area

*For information on this as on other points we are much indebted to Dr. Bergmann, Chief Surgeon of the Municipal Hospital, and his assistant, Dr. Kniazev.

in one of the healthy outlying districts of the town. The hospital consists of two spacious compounds, separated from each other by a public road. On one side of this street the main building is situated. The ground floor accommodates the female wards for (a) internal diseases and (b) gynecological cases, while the upper floor is occupied by the surgical department. The building is up to modern requirements and kept in excellent order; the principal drawbacks are that (a) it is often overfilled with patients and (b) there are no fly screens in the doors or windows. The kitchen for the whole hospital is housed in a separate newly erected building (costing \$9,000) situated near the main block. Across the street lies the male ward for internal diseases, which is of wooden construction. Next to this is a spacious isolation block for infectious cases. Lastly comes the newly erected ward for skin and venereal diseases.

From Table XII it can be seen that the outbreak among the hospital inmates began on August 11, while cholera cases had been admitted from outside since August 6. The latter were accommodated in one of the three sections of the isolation block; the two other sections were still occupied by typhoid and dysentery patients. On the first day of the hospital outbreak (August 11) 7 patients and 2 hospital employees (surgical nurse and janitor) fell sick simultaneously; of the 7, 3 were surgical patients (second floor of main building), 2 were on the ground floor, while the remaining 2 belonged to the male internal ward and the V. D. department, respectively. Two more cases occurred during the night (August 11-12), a third 24 hours later, a fourth on August 14—all among inmates of the main block.

One of the measures devised by the hospital authorities to determine the presence of carriers, if any, was to examine the stools of all patients accommodated in rooms where cholera victims had been living. This investigation was started on the morning of August 15 and yielded positive results in 6 of a total of 32 contacts (18.7 per cent). Of these, four were in the surgical ward (including a nurse), the other two being respectively in the male and female internal wards. It is an open question whether all the six were healthy carriers in the strict sense, as some of them had had gastrointestinal disturbances, usually slight diarrhoea. However, others, like the nurse, showed no signs of illness whatsoever.

A real cholera case was discovered on August 17 in a patient confined in the V. D. and skin ward.

There seems little doubt that at least the first eleven out of the fourteen cases contracted infection at one and the same time and probably from a common source.

Unfortunately, it was not possible to establish definitely the nature of this source. The first thought would naturally connect the outbreak with a contamination of the water supply; investiga-

tion, however, ruled this out. The hospital has a deep well which was found in excellent order; the water, when examined, was free from cholera vibriones. Regarding the food supply, the kitchen personnel yielded neither cholera cases nor carriers. No raw vegetables are given to the patients; all milk was boiled. The food was brought to the different wards by the kitchen staff in vessels belonging to the kitchen and distributed by them outside the wards; proper precautions were prescribed to avoid any contamination of the food containers through the crockery etc., of the infectious patients. The patients who contracted cholera had various forms of diet; there was nothing which all had in common.

Two theories may be conceived to explain the outbreak:

1. That it might have spread from the cholera patients previously admitted through (a) flies or (b) by intermediate persons (nurses or attendants). The hospital doctors disbelieve this and point out that among the patients suffering from other infectious diseases and still accommodated in the isolation block no cases occurred.
2. The hospital doctors claimed that the outbreak was due to the bread supply, which came from an outside bakery found to be rather insanitary. It was delivered by a cart hired each day for the purpose and not under the control of the baker. When the bakery was later inspected, one employee was missing and could not be found. The doctors alleged that this man might have had cholera. However, no proof was forthcoming for this. One important revelation was that not all the victims had bread on their diet list at the time.

After weighing the evidence, we are inclined to dismiss both the fly and the bread theory. When the first cholera cases were admitted into the Municipal Hospital, the personnel were not yet organized for epidemic work. Perhaps, either through inattention or overwork on the part of the nursing staff and lay helpers spread of infection through intermediate contact took place. WU LIEN-TEH, J. W. H. CHUN, R. POLLITZER.

TABLE I
Total Figures

Hospital	Admitted		Died		Mortality	
	Ch.	For.	Ch.	For.	Ch.	For.
Pinchiang	168	0	29	0	17.3	0.0
Municipal	26	40*	13	23*	50.0	57.5
Railway	32	23	9	10	28.1	43.5
TOTAL		226 63	51 33		22.6	52.4
		289	84		29.1	

*Including one Korean man.

TABLE II
Percentage Mortality

Pinchiang Hospital	17.3
Municipal Hospital	54.5
Railway Hospital	34.5

TABLE III
Sex Incidence

Hospital	Chinese				Foreigners				Total			
	Male		Female		Male		Female		Male		Female	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Pinchiang	135	80.3	33	19.7	0	0.0	0	0.0	135	80.3	33	19.7
Municipal	26	100.0	0	0.0	14	35.0	26	65.0	40	60.6	26	39.4
Railway	31	100.0	0	0.0	16	72.7	6	27.3	47*	88.7	6*	11.3
TOTAL	192	85.3	33	14.7	30	48.4	32	51.6	222	77.4	65	22.6

TABLE IV
Mortality Incidence As To Sex

(a) Pinchiang Hospital

		%
Male	25	86.2
Female	4	13.8
	—	—
	29	100.0

(b) Municipal Hospital

	%			%			%	
Chinese, Male	13	100.0	Foreign, Male	10	56.5	Total, Male	23	63.9
Chinese, Female	0	0.0	Foreign, Female	13	43.5	Total, Female	13	36.1
	13	100.0		23	100.0		36	100.0

(c) Railway Hospital

	%			%			%	
Chinese, Male	9	100.0	Foreign, Male	7	77.8	Total, Male	16	88.9
Chinese, Female	0	0.0	Foreign, Female	2	22.2	Total, Female	2	11.1
	9	100.0		9	100.0		18	100.0

(d) % Mortality in Males and Females Separately

HOSPITAL	CHIN.			
	CHIN. MALE	FEMALE	FOR. MALE	FOR. FEMALE
Pinchiang	18.5	12.1	0.0	0.0
Municipal	50.0	0.0	71.4	50.0
Railway	28.1	0.0	43.7	33.3

*Sex of two children unknown.

TABLE V

PINCHIANG HOSP.			Age Incidence				MUNICIPAL HOSPITAL			
CHINESE			CHINESE		FOREIGNERS		TOTAL			
Age	No.	%	No.	%	No.	%	No.	%		
0- 5	4	2.3	0	0.0	0	0.0	0	0.0	0	0.0
5-10	5	3.0	0	0.0	0	0.0	0	0.0	0	0.0
10-15	3	1.7	0	0.0	1	2.5	1	1.5	1	1.5
15-20	15	8.8	0	0.0	0	0.0	0	0.0	0	0.0
20-25	23	13.7	3	11.5	5	12.5	8	12.0	8	12.0
25-30	27	16.0	6	23.0	6	15.0	12	18.0	12	18.0
30-35	17	10.1	3	11.5	7	17.5	10	15.0	10	15.0
35-40	20	11.8	6	23.0	1	2.5	7	10.5	7	10.5
40-45	20	11.8	5	19.2	6	15.0	11	16.5	11	16.5
45-50	14	8.3	0	0.0	4	10.0	4	6.0	4	6.0
50-55	6	3.4	2	7.7	3	7.5	5	7.5	5	7.5
55-60	8	4.6	0	0.0	0	0.0	0	0.0	0	0.0
60-65	5	3.0	0	0.0	2	5.0	2	3.0	2	3.0
65-70	1	0.5	0	0.0	3	7.5	3	4.5	3	4.5
70-75	0	0.0	0	0.0	1	2.5	1	1.5	1	1.5
?	0	0.0	1	3.8	1	2.5	2	3.0	2	3.0
TOTAL 168 99.0			26 99.7		40 100.0		66 99.0			

TABLE VI

Age of Mortality Cases

AGE	PINCHIANG HOSPITAL		MUNICIPAL HOSPITAL				TOTAL	
	No.	%	CHINESE		FOREIGN		No.	%
			No.	%	No.	%		
0-5	1	3.4	0	0.0	0	0.0	0	0.0
5-10	0	0.0	0	0.0	0	0.0	0	0.0
10-15	0	0.0	0	0.0	1	4.3	1	2.8
15-20	0	0.0	0	0.0	0	0.0	0	0.0
20-25	5	17.0	1	7.7	3	13.0	4	11.1
25-30	3	10.2	2	15.4	4	17.4	6	16.6
30-35	3	10.2	2	15.4	2	8.7	4	11.1
35-40	3	10.2	5	38.5	1	4.3	6	16.6
40-45	3	10.2	2	15.4	3	13.0	5	13.9
45-50	5	17.0	0	0.0	2	8.7	2	5.5
50-55	0	0.0	0	0.0	2	8.7	2	5.5
55-60	4	13.6	0	0.0	0	0.0	0	0.0
60-65	2	6.8	0	0.0	1	4.3	1	2.8
65-70	0	0.0	0	0.0	3	13.0	3	8.3
?	0	0.0	1	7.7	1	4.3	2	5.5
	29	98.6	13	100.0	23	99.7	36	99.7

TABLE VII

Occupation

(a) *Pinchiang Hospital*

		%			%			%
Coolie	28	16.6	Mechanic	6	3.5	Student	2	1.2
No Occupation	27	16.0	Shoemaker	6	3.5	Prostitute	2	1.2
Housewife	27	16.0	Blacksmith	6	3.5	Cook	1	0.5
Small Merchant	16	9.5	Soldier	5	3.0	Bookseller	1	0.5
Servant	9	5.3	Tailor	5	3.0	Washerman	1.	0.5
Carpenter	9	5.3	Mason	4	2.3	Policeman	1	0.5
Ricksha Man	8	4.7	Printer	3	1.7	Coachman	1	0.5

168 98.80

(b) *Municipal Hospital*

	CHIN. MALE		FOREIGN MALE		FOREIGN FEM.		TOTAL	
	No.	%	No.	%	No.	%	No.	%
Out of work	2	7.7	5	35.5	8	30.4	15	22.5
Patients	0	0.0	2	14.2	10	38.0	12	18.0
Unknown	5	19.2	3	21.3	1	3.8	9	13.5
Housewife	0	0.0	0	0.0	5	19.0	5	7.5
Coolie	3	11.5	0	0.0	0	0.0	3	4.5
Police	3	11.5	0	0.0	0	0.0	3	4.5
Cook	2	7.7	0	0.0	0	0.0	2	3.0
Merchant	2	7.7	0	0.0	0	0.0	2	3.0
Prison Guard	1	3.8	0	0.0	0	0.0	1	1.5
Hospital Nurse	0	0.0	0	0.0	1	3.8	1	1.5
Hospital Janitor	0	0.0	1	7.1	0	0.0	1	1.5
Contractor	1	3.8	0	0.0	0	0.0	1	1.5
Carpenter	1	3.8	0	0.0	0	0.0	1	1.5
Servant	1	3.8	0	0.0	0	0.0	1	1.5
Midwife	0	0.0	0	0.0	1	3.8	1	1.5
Private Guard	1	3.8	0	0.0	0	0.0	1	1.5
Janitor	1	3.8	0	0.0	0	0.0	1	1.5
Cleaner	1	3.8	0	0.0	0	0.0	1	1.5
Old Cl. Merch.	1	3.8	0	0.0	0	0.0	1	1.5
Tailor	0	0.0	1	7.1	0	0.0	1	1.5
Sailor	0	0.0	1	7.1	0	0.0	1	1.5
Gardener	1	3.8	0	0.0	0	0.0	1	1.5
Teacher	0	0.0	1	7.1	0	0.0	1	1.5
TOTALS	26	98.5	14	99.4	26	98.8	66	99.0

TABLE VIII

Municipal Hospital

	TOTAL	DIED	% MORTALITY
Admitted from hospital	12	6	50.0
„ „ outside	52	28	53.8
	64	34	53.1

Note. The nurse and the doorkeeper who worked in the hospital but lived outside were not counted.

TABLE IX
DURATION OF STAY IN HOSPITAL

Days	Pinchiang Hospital		Municipal Hospital	
	No.	%	No.	%
1	22	13.0	13	19.9
2	35	20.8	7	10.7
3	28	16.6	4	6.1
4	23	13.7	2	3.1
5	22	13.0	3	4.6
6	15	8.8	5	7.7
7	6	3.5	2	3.1
8	6	3.5	2	3.1
9	6	3.5	2	3.1
10	2	1.2	2	3.1
11	0	0.0	4	6.1
12	1	0.5	1	1.5
13	1	0.5	3	4.6
14	0	0.0	0	0.0
15	1	0.5	1	1.5
16	—	—	2	3.1
17-19	—	—	0	0.0
20	—	—	1	1.5
21	—	—	2	3.1
22	—	—	3	4.6
23	—	—	1	1.5
24-25	—	—	0	0.0
26	—	—	3	4.6
27	—	—	1	1.5
28	—	—	1	1.5
	168	99.1	65	99.6

TABLE X
FATE OF PATIENTS ACCORDING TO THE DAY THEY WERE ADMITTED

Day of Illness	Pinchiang Hospital					Municipal Hospital				
	Admitted		Recov.	Died		Admitted		Recov.	Died	
	No.	%		No.	%	No.	%		No.	%
1st	32	37.4	27	5	15.6	16	24.2	7	9	56.3
2nd	33	38.6	25	8	24.2	19	28.7	11	8	42.1
3rd	11	12.9	9	2	18.1	11	16.6	4	7	63.6
4th	7	8.2	5	2	28.5	8	12.1	4	4	50.0
5th	1	1.2	1	0	0.0	8	12.1	3	5	62.5
6th	0	0.0	0	0	0.0	4	6.0	1	3	75.0
7th	1	1.2	1	0	0.0					
	85	99.8	68	17	20.0	66	99.7	30	36	54.5

TABLE XI

DIFFERENTIAL DIAGNOSIS (PINCHIANG HOSPITAL)

Acute Alcoholism	1
Gastroenteritis	4
Diarrhea	32
Dysentery	5
	—
	42

DETAILS OF MUNICIPAL HOSPITAL OUTBREAK

No.	Sex	Age	How Long in Hospital	Ward	Illness	Diet	Date Att. by Chol.	Result
1	F.	31	July 29	Surgic.	Appendic.	Common	Aug. 11	Died Aug. 12
2	M.	67	Aug. 2	Male's Intern.	Chron. Gastr.	Weak	„ 11	„ „ 11
3	F.	12	„ 3	Surgic.	Appendic.	„	„ 11	„ „ 12
4	F.	28	June 19	V. D.	Lues	Common	Aug. 11	Died Aug. 17
5	F.	70	Aug. 7	Women's Intern.	Pleuritis	Milk	„ 11	„ „ 13
6	F.	53	May 25	Surgic.	Ventral hernia	Common	„ 11	Recov.
7	F.	34	July 6	Women's Intern.	Morbus cordis	Milk	„ 11	„
8	F.	34	„ 22	Intern.	Pyelitis	Vegetable	„ 11- 12	„
9	M	34	Apr. 13	Surgic.	Tbc. & Amyloid	Common	„ 11- 12	Died Aug. 12
10	F.	43	Aug. 8	Gynecol.	Pelvieo- periton.	Strict	„ 12- 13	Recov.
11	F.	72	July 28	Surgic.	Fracture, Femur	Common	„ 14	Recov.
12	F.	61	Aug. 6	V. D.	Eczema helminth.	Common	„ 17	„
13	F.	40		Nurse, Surgic.			„ 11	Died Aug. 11
14	M.	30		Janitor, Main Bldg.			„ 11	„ „ 17

Remarks: Diets. Common—ordinary diet for convalescents.
 Weak—milk, jelly, toast, bouillon, eggs.
 Milk—milk, dishes made with milk, toast, or bread.
 Vegetable—vegetable, soup, milk, toast.
 Strict—milk, bouillon.
 The employees had common diet while on duty.

TABLE XIII

Died on Which Day of Illness

Day of Illness	Pinchiang Hospital		Municipal Hospital	
	No.	%	No.	%
1st	5	17.0	14	43.75
2nd	7	23.8	6	18.75
3rd	3	10.2	4	12.50
4th	3	10.2	2	6.25
5th	3	10.2	6	18.75
6th	2	6.8	—	—
7th	3	10.2	—	—
8th	0	0.0	—	—
9th	1	3.4	—	—
10th	0	0.0	—	—
11th	1	3.4	—	—
12th	0	0.0	—	—
13th	0	0.0	—	—
14th	1	3.4	—	—
	—	—	—	—
	29	98.6	32	100.0

TABLE XIV

Showing Instances of Successive Cholera Cases from the Same Address

	Address	Name	Sex	Age	Date of Admission
a.	Orphanage in 19th street	Sung	M	6	Aug. 10
		Tsia	M	8	„ 11
		Hsu	M	16	„ 12
		Li	M	14	„ 13
		Liu	M	14	„ 14
		Chang	M	50	„ 15
		Chen	M	57	„ 18
		Wang	M	60	„ 18
		Si	M	46	„ 18
		Liu	M	65	„ 20
		Chao	M	38	„ 21
		Chang	M	44	„ 21
		Chang	M	32	„ 21
		Chang	M	67	„ 21

b. Railway guard barracks	Chiao	M	42	Sept. 26
	Shu	M	31	„ 29
	Chao	M	32	„ 30
	Kuo	M	26	„ 30
	Lu	M	24	„ 4
c. 12th street, behind park	Tang	F	5	Aug. 14
	Tang	F	6 (sister)	„ 21
d. Harbin Municipal Hospital	See Table. XII			

B. Cholera Notes from Pinchiang Hospital, 1926

Introduction

Since the epidemic of cholera in 1919 when 4,500 deaths were reported in Harbin, we have been free from the visitation of this "tigerish" disease, though we heard of the occurrence of yearly cholera outbreaks in Shanghai.

This year cholera made its appearance in May in Shanghai and rapidly assumed a severe form, due in part to the exceedingly hot summer. The port was accordingly declared infected and quarantine measures were instituted at different seaports against it, including Tsingtau, Dairen, Newchwang, and Antung. In spite of all this, the infection was brought right through South Manchuria by carriers and the first recognized cases were seen at our hospital on August 5.

The beginning of the summer was exceedingly hot and dry; flies were in evidence in unusually large number, reminding us of the cholera year of 1919. We were fully prepared, therefore, against the invasion, having gained experience and proceeded to profit by it. Our hospital and staff have increased in equipment and personnel. Everything and everybody were ready to cope with any emergency.

A very large percentage of the patients (estimated at 90 per cent) came of their own accord and at an early stage of the disease for treatment, showing that the people of the city have heard of and have faith in our effective methods for dealing with this disease. Almost all of them were brought by one or more of their friends and relatives. Seventy-six per cent of them came to the hospital on the first or second day of illness, a large number of them having been acupunctured by native doctors in the basilic vein in one or both elbows. Though they were mostly brought by friends, our sanitary inspectors stationed at the four wards of the Chinese city have not been idle. At the time of the epidemic, their special attention was directed to the detection of cholera patients and their hospitalization, and it was partly due to their efforts that such a large proportion of the affected came. Looking at the map of the city, which we marked with little plugs to show the particular spot from which each

patient was hailed, one would be struck with the uniform scattering of the flags. They seemed to be almost evenly distributed throughout the whole city. Out of 168 true cholera cases, 30 came from the Railway Area, however.

Admission Routine

As soon as the patient was brought inside the compound, his friends were interrogated by the clerk as to particulars with regard to the name, age, occupation, day of illness, and address. Meanwhile, the doctor in charge examined the patient in the open air and determined whether the patient was suffering from cholera or not. If infusion was considered necessary, he was at once carried into the infusion room where eight patients could be injected at the same time. The doctor or dresser in charge gave the injection while another dresser sat by the patient and kept watch to see that the arm did not move and that the saline was running in properly. After infusion was completed the patient was carried to block No. 1 where new cases were admitted. A label with a number was tied to the wrist for identification; his name and number were put on the door of the room. When block No. 1 was full, the older cases were transferred to block No. 2.

The criterion for the need for infusion was the pulse. If it was small or impalpable, then saline was indicated. On the other hand, if it was good, and the general appearance of the patient warranted, only kaolin water was given and the patient put in a cubicle in block No. 1. Often it was possible to make a correct diagnosis at a glance, but if there was any doubt, the patient was made to pass a stool in an earthenware bowl and the stool was examined by the bacteriologist. The important point is that no time was wasted in unnecessary formality. As soon as the patient arrived, he was given attention. This aspect deserves special mention, because the time element is very important and delay may turn the scale against the chances of recovery.

We found it sufficient in dealing with this epidemic to maintain two doctors on day duty and one at night. Three shifts of dressers were arranged for the twenty-four hours at eight hours each, so that patients were attended to at all times of the day and night. We also had two shifts of day and night attendants. In this way new cases were promptly treated, and old cases were watched all the time. If their conditions demanded, a second and a third injection could be given, or other auxiliary therapeutic measures administered. We prepared hypertonic saline solution in 1,000 c.c. flasks and kept them warm all the time in a large water tank heated with

firewood in a room in the compound. In this way, warm sterilized saline could be had at any time of the day or night.

A small temporary laboratory was fitted up near the wards where the examination of urine and other simple tests could be carried out. The stools were examined and cultivated in the big laboratory in another compound.

Clinical Picture

All particulars of the patients, such as age, occupation, sex, etc., are embodied in the form of tables. Suffice it to say that all the patients were of Chinese nationality; 80.3 per cent were males, the rest, 19.7 per cent, were females, as against 92.8 per cent males and 7.2 per cent females in 1919; as before, young adults between 25-30 years of age formed the highest percentage of patients; coolies, persons of no occupation, and housewives were among the commonest of the patients; most of them stayed from one to five days.

Out of 168 admissions, 29 died, a mortality of 17.2 per cent. Among these 29 deaths, 25 (86.2 per cent) were males and 4 (13.8 per cent) were females. The highest mortality was shared equally between those at ages 20-25 and 45-50 (17 per cent). The largest number of patients died on the second day of illness (23 per cent), showing that the collapse stage was the most dangerous. After the seventh day of illness, the chances of recovery were good, for very few died if they could survive up to this period. The chief cause of death may be said to be collapse; some severe cases or cases weakened in any way by previous conditions died in spite of every effort to save them. The second danger was uræmia; the third, heart failure; the fourth, toxæmia; and the fifth, pneumonia. We had two cases with delirium—one on the second day of illness and the other on the fifth day. The prognosis for these cases seemed very bad. The latter, a strong young man, died in the hospital, and at post mortem his organs showed signs of chronic alcoholism.

Reviewing the cases, we may say on the whole that they were on the light side. Suppression of urine caused anxiety in some cases, while albuminuria was very frequent. A few died of uræmia, notably an old man of sixty-three, who stayed in the hospital for fourteen days. His urine showed much albumen and acetone. On the fourth day of stay, he developed coma, which was successfully treated by venesection, 300 c.c. blood being withdrawn. He gradually improved under protoclysis with normal saline, but died of hypostatic pneumonia.

Now a word about toxæmia. Patients almost always showed a reactionary fever after infusion, the rectal temperature being

the best guide. But when the purging stopped either naturally or as the result of the hypertonic infusion, absorption of cholera toxin from the intestines caused a rise of temperature; often 103° F. was registered. Two cases showed signs of toxæmia, one a strong young man of 31, who was treated on the first day of illness with infusion. On the second day he seemed better. On the fourth day, however, he showed high fever, was unconscious, and a well-marked septic rash appeared on his trunk and thighs; death occurred on the next day. The other was a smallish man of 27, who was injected twice, 3,000 c.c. being given on the first day and 2,000 c.c. on the second day. It was seen also that he suffered from a tertiary syphilide. On the seventh day, he developed fever and hiccough. His urine was repeatedly examined; only a trace of albumen was found and no acetone. A marked septic rash appeared all over his body. Though his condition was critical, he gradually improved and left the hospital on the fourteenth day.

Acidosis was often met with, as indicated by the presence of acetone in the urine. For the treatment of this condition, Sir Leonard Rogers advocated the routine administration of large doses of sodium bicarbonate (gr. 160 to a pint of normal saline), one pint being injected intravenously just before the hypertonic saline infusion. Owing to the difficulty of sterilizing the bicarbonate, because heat changes the bicarbonate to carbonate which causes necrosis to the tissues unless the solution is injected straight into the vein, we have not adopted his method, but relied on the administration of large doses of sodium bicarbonate by the mouth.

The typical textbook picture of a cholera patient need not be described here. The severe or moderately severe cases can be easily recognized. The incipient or light cases have to be admitted and watched. Their physical condition and their stools must be examined. Not all of them voided the typical rice-water stool, for again and again the bacteriologist was able to isolate the vibrio cholerae from yellow stools, or bile-stained or even blood-stained stools. The final diagnosis rested with the isolation of the vibrio and the agglutination by specific serum.

Mention may be made shortly of the different other diseases which we had to diagnose, viz., drunkenness, gastroenteritis, summer diarrhoea, dysenteries, typhoid, and paratyphoid. In case of doubt and before a definite diagnosis could be made, we administered the hypertonic saline infusion, if the physical condition, the pulse, and the blood pressure of the patient demanded it.

Treatment

Prophylaxis. Warning was given to the public in the form of bulletins and newspaper articles against cholera. Personal prophylaxis in the form of anticholera inoculations was advocated and practiced. The means of spread of the disease was carefully explained and blame was put on flies, soiled fruits, vegetables, and uncooked food. The police helped by the prohibition of the sale of melons, which was then in season.

In order to protect our staff against cholera, they were inoculated with the anticholera vaccine which was manufactured in our own laboratory. Before they entered the cholera ward, overalls were donned, and rubber galoshes were put over their shoes. On emerging, they rubbed the soles of the galoshes on doormats soaked in izal fluid which were placed in front of the doors. The hands were soaked in a basin of the disinfectant placed outside the wards. They were instructed to wash their hands very carefully before taking food. When they had to soil their hands in the course of their work, they wore rubber gloves. That these and other precautions were efficient was shown by the fact that none of our staff got infected.

For the accommodation of the cholera patients, plain wooden beds were used, with no mattress. By the bedside an earthenware basin containing a little disinfectant fluid was placed to receive the vomitus, stools, and urine. Flies were excluded by window and door screens. Fly powder and fly paper were also freely used. The stools were emptied into barrels containing disinfectant and they in turn were emptied into a covered pit as they became full.

The food of the patients consisted only of congee (rice broth). As they improved, gradually other items were added, such as soup, biscuits, and plain Chinese cakes. Great difficulty was experienced in combating the natural craving for more food and the smuggling of unsuitable eatables by friends.

Therapeutic Measures. We relied mainly on the hypertonic saline intravenous infusions as a means to restore salts and fluid in the system. Often the patient recovered immediately and it would appear as if snatched from the grave. In the collapse stage there does not appear to be anything superior. We used the ordinary douche can and a six-foot rubber tubing. A 1-c.c. syringe barrel was fitted to the other end of the tube to which an intravenous needle, as large as convenient, was attached. The basilic vein at the elbow was usually selected and made to stand out by constriction of the

arm; the skin was cleansed with spirit and the needle plunged into the vein. If blood flowed into the glass barrel, then the saline was allowed to run. Usually 1,500 c.c. to 3,000 c.c. were injected at one time and as rapidly as possible. Generally the injection was completed in half an hour. The saline was given at about 104° F. in the douche can. The condition of the patient was watched. If the pulse was full and bounding or if the shivering or restlessness was excessive, the injection was stopped.

Some patients required one injection; most patients required two, not necessarily on the same day; some required three or more; while others could not be saved by any means. If the vein could not be entered, we dissected it out and injected by the open method or the saline was given subcutaneously. Coming to auxiliary measures, mention must be made of kaolin. For light cases and also for moderately severe cases after infusion, we gave a bowlful at a time every two hours; in all, if possible, six bowlfuls were given. The kaolin was suspended in water, 100 grams in 250 c.c. We found some of the patients could not tolerate it well, because they almost always vomited directly they swallowed it into the stomach.

Similarly, weak potassium permanganate solution, 1 in 2,000, was freely given, but it was not liked by some of the patients.

For the failing heart we used hypodermic injections of digitalin, camphor, and adrenaline. These auxiliary measures were highly useful, when one considers the real cause of the collapse. It is true that the withdrawal of fluid and salts from the system plays a great part, yet the cholera toxin exerts a very great influence in the production of the well-known clinical picture, namely, sunken eyes, anxious facies, cold extremities, hoarse voice, cramps, and weakened heart. Both kaolin and permanganate tend to neutralize the toxin manufactured by the cholera vibrio in the intestines. We consider them therefore exceedingly useful adjuvants. We have not used any anticholera serum, and so cannot express any opinion on its merits.

For acidosis, we used large doses of sodium bicarbonate per os as already stated.

For the treatment of uræmia, we believe in subcutaneous injections of physiological saline, or proctoclysis together with cardiac stimulants. If coma supervened, we employed venesection.

Cholera is a disease which has tigerish characteristics, as the Chinese consider it. It attacks suddenly, but it also leaves

the patient well quickly. In treating this disease, the physician demonstrates exceedingly clearly the great principle of medicine, namely to assist nature and not harm the patient. For instance, he must not check the purging by the administration of opium; rather he must replenish the lost fluid and salts, neutralize the toxin, stimulate the heart, and treat each complication as it makes its appearance. Given a fair chance, nature will do the rest, if the attack is not overwhelming and the patient is not weakened by previous illness, or indulgence. The physician, on his part, must act quickly, observe closely, and be ready to meet all emergencies, exemplifying the qualities of which Henley wrote:

Faultless patience and unyielding will,
Beautiful gentleness and splendid skill.

Formula of Hypertonic Saline Solution

Sodium chloride	gr. 120	(8	grams)
Calcium chloride	gr. 4	(0.25	„)
Potassium chloride	gr. 6	(0.40	„)
Distilled Water	pint 1	(568	c.c.)

C. Laboratory Reports

In view of the fact that in some parts of China no advantage is taken of bacteriological methods for the diagnosis and control of cholera epidemics, it may be well to give some hints as to the simple means we used in our work. We performed this usually with two media only, peptone solution and ordinary nutrient agar of alkaline reaction. The use of special media, in the first line of Dieudonné Agar (3 per cent neutral agar mixed with defibrinated and alkalized cattle blood in a proportion of 7:3) is very advantageous but can be dispensed with for routine work; we used this—apart from scientific investigations—only in special cases.

We prepared peptone solution according to the formula contained in the German “Anweisung zur Bekaempfung der Cholera”:¹

Take	Peptonum siccum Witte	100.0	gram
	Sodium chloride	100.0	„
	Sodium nitrate	1.0	„
	Cryst. sodium carbon	2.0	„
	Sterile distilled water	1,000.0	c. c.

¹ Quoted by Klimmer, *Technik & Methodik der Bakteriologie & Serologie*, Berlin, 1923, p. 8 and following.

Mix solids with water and dissolve by heating. Then filter (if necessary twice through same paper), fill in flasks of 100 c.c., and sterilize in the autoclave. When peptone solution is wanted, one part of this stock solution is mixed with 9 parts of sterile distilled water, filtered if necessary, filled in small flasks or test tubes as wanted. Sterilize in the autoclave.

1. Examination of Stools

From the material (a) films and (b) cultures in peptone solution are made. In early suspicious cases 1-3 flasks containing 50 c.c. of the solution are used; during an epidemic it is sufficient to use test tubes containing 10 c.c. of the medium if small quantities of the feces are taken, a mucous floccule being picked out if present.

(a) The films are stained with freshly diluted carbol fuchsin (1:10). Sometimes it is impossible to come to a *prima-facie* diagnosis by inspections of the films. On one hand, as was our experience in the 1926 epidemic, only a few characteristic vibriones may be present in addition to other bacteria, though good cholera cultures are easily obtained from such material; on the other hand, in many noncholeraic stools forms resembling the *vibrio cholerae* may be seen which may prove misleading if caution is not used. At the best, not more than a suspicion of cholera can be reached by inspection of smears.

(b) The inoculated flasks or tubes are incubated for three to five hours; in early cases it is well to examine them both after three and five hours; during an epidemic and in cases with typical stools we can with safety proceed after three hours.

No reliance should be placed upon the mere appearance of peptone cultures. It is often maintained that—if cholera vibriones are present—the cultures become—owing to the rapid growth of this kind—cloudy after three hours' incubation, whilst clearness of the fluid practically excludes cholera. In this epidemic we have seen instances of cloudiness after three hours in spite of the absence of cholera vibriones; on the other hand, the cultures may be almost or even quite clear after five hours, though good growths of cholera bacilli are obtained from them; the latter category of cases is rare.

The methods chosen for examination and further treatment of the peptone cultures vary according as to whether (i) no great dispatch is necessary (clinically typical cases during an

epidemic, contacts, etc.) or (ii) a speedy diagnosis is wanted (early, atypical cases, etc.).

(i) In the first case it is sufficient to take with the platinum needle one drop from the upmost part of the peptone culture (gently tilting the vessel if a membrane has formed on the surface to avoid touching this) and to place this drop on an agar dish. This is then spread out with the aid of a sterile glass spatula and a second dish inoculated with the same spatula. After twelve hours good growth will be obtained. Cholera colonies show, when inspected from above, a grayish-white color, whilst in transparent light they assume a distinct bluish luster. This is well seen when standing not quite near but at some distance from the window and enables one to detect colonies from which smears are to be made. When cholera-like bacilli are seen, such colonies may either be directly transplanted on agar slants or they may be passed through peptone water and then on slants. The last method is more certain to give a pure culture for agglutination tests.

(ii) The procedure to be chosen when dispatch is necessary depends upon an inspection of films taken from the topmost part of the peptone cultures. If vibriones are found in pure culture, then it may be possible to proceed directly with agglutination tests. Often this will not be the case; then two methods have to be applied:

1. New peptone flasks or tubes are inoculated under the precautions described above (after three and five hours' incubation). This may still be repeated one or several times at three hours' interval until a pure culture of vibriones is obtained.

2. Agar dishes are inoculated after three and five hours' incubation from the original peptone cultures or from one of the subcultures where a fair number of vibriones is seen .

If cholera is present, it will usually be possible to obtain a pure culture after three to eighteen hours.

The technic of the agglutination tests is described in all textbooks on bacteriology and serology. Either the microscopic or macroscopic methods may be chosen. It is necessary to make controls with a proved cholera strain and with suspensions of both this and the bacteria to be tested in physiological salt solution to watch for any tendency to sedimentate spontaneously. If quick results are desired and a peptone culture showing pure or almost pure growth of vibriones has been obtained, a good preliminary test may be made by simply

adding with a capillary pipette one drop of the agglutinating serum. This corresponds to a dilution of 1:200 to 1:500 and the positive result (which is seen within half an hour) establishes strong *prima-facie* evidence. By this method it is sometimes possible to come to a decision within four hours.¹

At the height of a bacteriologically established epidemic it may not be necessary to test each strain by agglutination. If only few workers are available bacteriological examination for every case may not be necessary, since the time is better spent upon more urgent tasks, such as preparation of vaccine, examination for carriers, of water, etc. But bacteriological and serological tests are essential in nontypical cases.

A few words may be devoted to the mass examination of feces for detection of carriers needed sometimes for maritime or railway quarantine work. In this connection the method adopted by the Japanese authorities at Dairen may be recommended.

This consists, in the case of steamers arriving in port, of

1. Enforced defecation by all members of crew and passengers on the morning of arrival and collection of small samples in little Petri dishes.

2. Placing of a small particle of specimen with sterile toothpick in test tube containing peptone water.

3. Handing over of all the peptone cultures (4-6 hours old) in racks of thirty tubes each to the quarantine authorities.

4. Making of films (five on one slide), drying over a spirit lamp, staining with dilute carbol fuchsin for $\frac{1}{2}$ min., drying between filter paper without washing in water, then examining under the microscope.²

After this introduction we can discuss the findings made in the past epidemic.

1. Examination of Stools

a. Patients. It is essential for the laboratory workers to be always in close touch with the clinicians so as not to interpret negative findings wrongly. Owing to the presence

1. If agglutinating serum cannot be prepared at the spot, it is easily procured from one of the serological institutes in China.

2. In this way, Dr. Yano, of the Naval Quarantine Service, could, with the aid of another trained doctor assistant inspect six slides, that is, 30 specimens, in one minute. While I was in the Laboratory the examination of 280 specimens took just 40 minutes from the time of arrival of the peptone cultures to the final report by telephone to the shipping authorities. In addition to Dr. Yano, there were two medical assistants and six technicians in August, 1926, undertaking these fecal examinations for steamers arriving from Shanghai (W. L. T.)

of many other microorganisms in the feces or to other untoward circumstances, it may happen that no cholera growths are obtained. If the case is clinically suspicious, new stool samples must be procured and only after two or three examinations are proved negative can the patient be declared cholera-free though clinically suspicious. We saw a few such instances where the first examination was negative, but the second positive.

Only in twenty-five per cent of the positive cases the stools were typical, rice-water-like. In the rest they were liquid, often with mucus, and more or less stained. In one instance (Pt. 199) the stool was of a more pasty consistence and showed many gas bubbles. Patients 29 and 201 voided pinkish but otherwise typical stools. Patient 186 had distinct admixture of blood in the stools; the findings in his case were as follows:

Sept. 3: Stool consists of a watery pink liquid in which white and reddish floccules are suspended. Cultures show growth of somewhat atypically shaped vibriones; agglutination positive.

Sept. 4: Stool as yesterday; in unstained specimens red blood corpuscles and many leucocytes were seen, but no amœbæ. Cultures negative for cholera.

Sept. 5: Typical, rice-water-like stool, containing no blood. Culturally, cholera positive.

Presumably the admixture of blood in these cases was due to some preëxisting process (internal hæmorrhoidal nodes, etc.).

b. Personnel. No positive findings were obtained. This is not surprising, as all precautions, including vaccination, were taken.

c. Contacts. Among the relatives or friends of the patients positive findings proved by agglutination were obtained in 2 out of 43 cases, i.e., in 5 per cent. Probably the percentage would have been greater, if the stool of each contact had been examined not once but repeatedly; this, however, was only rarely possible. The following data are given regarding the two "carriers":

Case A was a strong young man, contact to our No. 61 cholera case, admitted August 18, died August 21. From this contact on August 20 a sample of liquid brown stool was obtained. Smears showed only a few suspicious forms, but a pure culture of vibriones, giving a positive agglutination test,

was easily obtained. When seen on August 21, the man declared himself to be quite well; he was just eating raw apples! Normal pulse and temperature. He said that he had slight diarrhoea a few days before, but only one stool each of the last two days. On the twenty-second and twenty-third he continued well, but had two liquid stools daily. Then he swallowed of his own accord opium in order to cure himself. A swab taken on the twenty-third still gave a positive result. On the twenty-fourth the stool was of normal consistence, brown in color. Suspicious forms were seen in films and positive cultures were obtained.

This was probably a light ambulant case and not a carrier in the strict sense.

Case B, in contact to patient No. 73 (admitted on August 20), had on August 23 a pasty, almost liquid stool, yellow in color, resembling that of a healthy infant. A pure culture, showing positive agglutination, was obtained after some sub-culturing. Unfortunately the man disappeared soon after the material was taken so that no history or further samples could be obtained. As far as could be established, he was well.

On one occasion cholera-like germs were obtained. This was from a contact to our No. 127 cholera case, admitted on August 24. From a solid brown stool voided by the contact on August 24, bacilli resembling cholera vibriones morphologically and culturally were obtained; repeated agglutination proved negative.¹

II. Examination of Water

We found the technic recommended by the German regulations (1) useful: 1,000 c.c. water to be examined were collected in a large sterile flask. To this 100 c.c. of the peptone stock solution were added and the contents after thorough shaking distributed in small flasks of 100 c.c. Examination after an incubation of 8 and 24 hrs. Films were taken and the flasks which showed suspicious growths were proceeded with as in the case of stools.

No positive results were obtained from two houses, in each of which several cholera cases had occurred (one examined

1. A strain somewhat resembling cholera bacilli was obtained also from patient No. 206 on September 13 from liquid stool showing many mucous floc-cules, some of which were blood-stained. Clinical diagnosis: Dysentery. The bacilli in question were not typical in appearance, being more crescent-shaped with somewhat pointed ends and showed nonluxuriant growth. So the negative agglutination test merely confirmed our conviction that we were not in the presence of cholera. On the next day typical dysenteric stool, containing neither cholera-like germs nor amoebae. Diagnosis: Bacillary dysentery.

twice), or from the river Sungari. However, from the river water we obtained on two occasions bacteria much resembling the cholera vibrio. Repeated agglutination tests proved negative. A classification of these germs and those obtained from the contact has not yet been concluded.

III. Examination of Urine

The findings of albumen and acetone are shown in the table. The samples were almost all pale in color and of low specific gravity. Reaction uniformly acid. Even when only traces of albumen were present, usually a number of renal casts and also renal epithelia were seen in the deposit. Blood was once found; the woman in question was possibly menstruating. Diazo reaction was negative in several cases tested. Indican was only rarely slightly augmented, chlorides almost always normal in quantity.

IV. Post-Mortem Examinations

Altogether we performed fourteen *post mortems* upon individuals in whom cholera had been diagnosed or suspected. Of these, eight proved positive; one, suspicious; five, negative.

Little need be said about the negative cases. The two dissected in our hospital both showed purulent peritonitis due to dysenteric ulcers of the intestine. Of the three seen at the Municipal Hospital the first had a chronic cholecystitis and cholelithiasis; death was apparently due to the passing of a gallstone, the diseased heart of the patient (lipomatosis and myodegeneration due to atheroma) being unable to overcome this crisis. The second showed croupous pneumonia, leading to septicæmia and colitis. The third patient was an old man with emphysema, chronic bronchitis and bronchiectatic cavities, and myodegeneration of the heart, who could not withstand an attack of subacute gastroenteritis. Investigation for cholera vibriones proved negative in his case.

The records of the cholera cases, including a suggestive but bacteriologically negative one, are as follows:

(a) *Pinchiang Hospital:*

CLIN. NO.	SEX.	AGE	DATE	EXTRACT FROM CLINIC. HISTORY	IMPORTANT P.-M. FINDINGS
1	1	M. 59	Aug. 7	Admitted Aug. 6 with typical cholera symptoms. Died in the col-	Permission obtained for partial p.m. only. Ileum shows typical cholera lesions. Severe enteritis

N.B. In every case except No. 4 the diagnosis of Cholera was confirmed by bacteriological tests.

CLIN. NO.	SEX.	AGE	DATE	EXTRACT FROM CLINIC. HISTORY	IMPORTANT P.-M. FINDINGS
				lapse stage on forenoon of Aug. 7.	in colon. Severe de- generation of kidneys. Spleen congested.
2 37	M.	61	Aug. 16	Admitted with severe typical symptoms on Aug. 14, when ill for 3-4 days. Rec. three infusions, yet could not overcome collapse stage. Very mark- ed albuminuria ($\frac{1}{2}$ Vol.)	Emphysema and hypostat pneumonia. Lipomatosis and paren- chymatous degeneration of heart muscle. Fatty degeneration of liver. Spleen not enlarged but somewhat soft and congested. Acute parenchymatous neph- ritis. Many submucous hemorrhages in sto- mach. Serosa of ileum injected in parts; mucosa changes also only at spots, elsewhere the mucosa not swollen and rather pale. Con- tents liquid, stained. Colon free. In rectum submucous hemorrhages and some ulcers, app. chronic in nature .
3 56	M.	25	Aug. 24	Admitted Aug. 18 with severe typical symptoms. 3 in- fusions. Somewhat better next day but had to be injected again on Aug. 20. Diarrhea and vomiting then stopped. On Aug. 23 delirium set in and sym- ptoms of pneu- monia (bloody ex- pect.) became	Extensive confluent lo- bular pneumonia in both lower lobes; foci also in upper lobes; acute emphysema. Hypertrophy of left ventricle of heart; myodegeneration. Liver rather hard, shows at places fatty degenera- tion. Spleen somewhat soft, not congested. Large white kidney. Pancreas indurated. Bladder contains urine.

CLIN. NO.	SEX.	AGE	DATE	EXTRACT FROM CLINIC. HISTORY	IMPORTANT P.-M. FINDINGS
				manifest. Died same day. Was apparently a drinker.	Serosa of ileum shows slight injection at places, mucosa extensive inflammation. Contents yellowish brown with admixture of mucous floccules. Colon not changed with greenish liquid contents. Stomach almost empty; injection of submucous vessels. This was apparently a drinker suffering from chronic parenchymatous nephritis.
(b) <i>Municipal Hospital:</i>					
4	4 M.	25	July 27	Had left Shanghai about 5 days ago. Fell sick during journey with vomiting and diarrhea. Sent from station to Hospital where he seemed rather suspicious for cholera. Bact. exam. negative. Adm. July 20, died 26th with symptoms of uræmia.	Acute hemorrhagic gastroenteritis, specially of ileum and colon. Submucous hemorrhages in stomach. Hypostatic pneumonia. Lung œdema. Acute nephritis. Parenchymatous or early fatty degeneration of the organs. Examination of intestinal contents negative for cholera.
5	16 F.	40	Aug. 12	Nurse of surgical ward. Collapsed suddenly with vomiting and diarrhea on Aug. 11, died same day.	Partial post mortem shows typical appearances in the small intestine. Stomach and colon free. Parenchymatous or early fatty degeneration of organs.

CLIN. NO.	SEX.	AGE	DATE	EXTRACT FROM CLINIC. HISTORY	IMPORTANT P.-M. FINDINGS
6 8	M.	67	Aug. 12	Patient admitted to internal ward of Hosp. on Aug. 2 for chronic gastritis. Cholera history same as in Case 5.	Partial post mortem. Only moderate injection of ileum serosa. Small intestines seem somewhat contracted. Severe desquamative enteritis in ileum. Other organs not seen.
7 13	M.	25	Aug. 13	Admitted on Aug. 8 with typical cholera symptoms. Died on 11th, evidently of uræmia.	Partial post mortem. Serosa of intestine only moderately injected; also the mucosa shows severe catarrh only at places. Contents liquid, yellowish. Submucous hemorrhages in stomach. Acute parenchymatous nephritis. Fatty degeneration of liver. Spleen congested and soft.
8 1	M.	34	Aug. 13	Patient admitted on April 13 for tbc. of lungs and intestine. Developed cholera symptoms on Aug. 12, died same day.	Chronic tbc. of lungs. Tbc. ulcers of intestines. Amyloidosis of liver, spleen, and kidneys. Ileum almost free. Typical rice-water cont.
9 57	M.	41	Sept. 8	Sailor, admitted with typical symptoms on Sept. 5 on 4th day of illness. Drinker. When admitted severe symptoms and typical stools. Later on stool liquid, <i>bloody</i> . Could not over-	Partial post mortem. Chronic catarrh of stomach. Submucous hemorrhages near the pylorus. Serosa of ileum only partly injected. Acute desquamative catarrh in ileum. Enteritis in jejunum. Submucous hemorrhages both in

CLIN. NO.	SEX.	AGE	DATE	EXTRACT FROM CLINIC. HISTORY	IMPORTANT
					P.-M. FINDINGS
				come collapse stage and died on Sept. 6.	jejunum and ileum. Severe hemorrhagic in- flammation of the des- cendent colon and sig- moid. Fatty degen. of liver. Spleen somewhat swollen and soft, con- gested. Acute parenchy- matous nephritis.

There is no need to enter here in a discussion of the well-known morbid changes found at *post mortem* in cholera cases. What we wish to stress is the great variability of the essential signs, even in cases succumbing quite early in the disease. Our cases Nos. 1 and 5 alone may be said to have exhibited all the classical features, though in the former the simultaneous affection of the large intestine has to be noted. The other cases, though most often succumbing in the collapse stage, were in one way or the other not so well marked. In most of these the findings in the small intestine as well as the acute changes in the kidneys and the general signs (marked *rigor mortis*, dryness of the tissues, emptiness of the bladder, etc.) would have left little doubt as to their cholera nature, even if their history had not been known. If, however, cholera intervenes in individuals already suffering from certain diseases, the diagnostic task confronting the pathologist might, indeed, become difficult. This holds true of our case No. 8. This man suffered from amyloidosis as well as lung and intestinal tuberculosis. It is true that in his case the kidneys were softer and more hyperæmic than is usual in such cases, but this was not conspicuous. The rice-water stools were characteristic, but the complete absence of acute changes in the intestinal wall might suggest otherwise. Case No. 3, showing chronic nephritis, could not have been misinterpreted because of the presence of intensive inflammation in the ileum and signs of desquamation (plentiful admixture of mucous floccules in the liquid, yellowish-brown stools).

In addition to the small intestine, especially the distal portion, changes may be found in parts of the large intestines. These were either due to the same acute cholera infection or were already existent. The ætiology of the severe hemorrhagic

inflammation found in Case 9 in the descending colon and sigmoid is not yet histologically elucidated. These changes, though seemingly acute, are in all probability not *directly* caused by the cholera infection. The individual in question was a drinker who, while ill at home for three days, tried to cure himself with strong alcoholic concoctions.

The inflammatory changes found in the large intestine as well as the submucous hemorrhages in the stomach and intestine in several cases (2, 7, 9) attracted our special attention as they suggested the cholera nature of Case No. 4, observed some time before the presence of the epidemic in Harbin was fully established. At the time we performed autopsy (No. 4) we were skeptical as to its cholera nature owing to the decidedly hemorrhagic lesions in the ileum, the cæcum, and the ascending colon. Everywhere the mucosa was suffused with blood. Later on, when comparing the findings of this case with those in established cholera cases succumbing some time after the onset, we became rather suspicious, though three bacteriological examinations had proved negative, and the serum of the patient had failed to agglutinate cholera bacilli.

V. Preparation of Vaccines

During the epidemic our laboratory prepared from local strains over 60,000 doses of anticholera vaccine for distribution among employees of the railway, municipality, police, soldiers, students, and other inhabitants in the district. Supplies were also sent to outstation hospitals, including Newchwang. Judging by the number of persons receiving prophylactic inoculations we feel that the public has come to appreciate this means of prevention. We found the reaction in all cases slight, and, as far as we know, no person who had received inoculation developed cholera.

Summary of Laboratory Findings

I. Examination of Stools

	<i>Total</i>	<i>Positive</i>	<i>Negative</i>
a. Patients	40*	28	12
b. Personnel	14	0	14
c. Contacts	43	2	41† (5% positive)

II. Examination of Water

a. From houses	3	0	3
b. From river	2	0	2‡

* In a number of other cases examinations were made by the clinicians.

† Once cholera-like germs obtained.

‡ On both occasions cholera-like germs isolated.

*III. Examination of Urine**

	<i>Total</i>	<i>Positive</i>	<i>Traces</i>	<i>Negative</i>
Albumen	45	20	13	12
Acetone	45	4	0	41

IV. Post-Mortem Examinations

	<i>Total</i>	<i>Positive</i>	<i>Suspicious</i>	<i>Negative</i>
	14	8	1	5

R. POLLITZER.

* In a number of cases examinations were made by the clinicians.

INVESTIGATIONS ON THE VITALITY OF VIBRIO CHOLERAE ON CHINESE PAPER MONEY

BY H. M. JETTMAR

It is well known that the vibrio cholerae when dried on solid objects dies off in a comparatively short time. Numerous investigations, mostly experimental, dealing mainly with the vitality of vibrio cholerae on solid victuals confirm the fact. Of course, the small degree of moisture frequently present on the surface of such objects may delay considerably its death.

Thus, these microorganisms may be found alive on the cut surfaces of *fruits* for days or even weeks. Melons seem even to be a good nutrient medium for them (1), if daylight and intensive exsiccation are avoided. Pollak (2), investigating the vitality of vibrio cholerae on different kinds of vegetables and fruits found that these microorganisms were only slightly damaged by diffuse daylight. Especially on the inner leaves of cabbage the bacilli remained alive for a long time (several weeks). Thus the author comes to the conclusion that although the conditions of these experiments were artificial (the victuals being plunged into an emulsion of a pure culture of vibrio cholerae), the results emphasise the risks, which may attend the sale or consumption of fruits and vegetables coming from cholera-infected quarters.

T. P. Mackie & G. Trasler (3) stated that vibrio cholerae smeared over slices of melons remained alive for more than one day.

The vibriones may be found alive at least one day on slides of fresh unwrapped rye *bread* when exposed to the air, up to three days, if the bread was rolled up in paper, and more than a week if laid under a glass shade thus preventing exsiccation (4). Under ordinary conditions cholera vibriones are not alive on bread for more than eighteen hours, according to Bruni (5).

Friedrich (1) observed the decaying of vibrio cholerae on *pastries* at most after 24 hours, only on biscuits they remained alive from 1-4 days.

On damp *clothes* the Cholera bacilli remain alive for a considerable time; on the surface of damp linen they may even intensively proliferate (6).

According to Gamaleia (7), Hesse (8), Berckholtz (9), Germano (10) the spirilla dried on clothes, may live from 1-5 days.

On absolutely *dry surfaces* exposed to intensive exsiccation the spirilla die after a few hours.

Cholera vibriones dried on *glass* are already dead after two hours (5). When intensively exsiccated or exposed to direct sunlight they perish even sooner.

Cholera vibriones dried on *silk* threads in the exsiccator are dead after 2-4 days (12).

When kept on dry *tobacco* and *cigars* the cholera spirilla die very quickly, at most after 7 hours (1). According to Wernicke (13) the vibriones die even more quickly on these objects than on cover glasses. Even on moist cigars and snuff they perish within 24 hours.

The vitality of vibrio cholerae on *paper sheets* is likewise a short one. According to Uffelmann (4) cholera vibriones dried on printing paper were alive for at least 17 hours if kept in a closed book, at least for 23½ hours, if wrapped in an envelope, and if kept on postcards they lived for 20 hours.

It is of practical interest to know, how long vibrio cholerae may live on coins and banknotes.

The vitality of the vibrio cholerae on coins has been already studied. Uffelmann (4) stated that the cholera vibriones perish on coins very quickly, already after 10-30 minutes. On silver or copper coins they disappear—apparently on account of some chemical influences—much quicker than on gold coins.

The vitality of cholera vibriones on *banknotes* has not yet been systematically investigated so far as I know.

This, however, is a question of some importance for North China with its almost exclusive paper currency. Therefore, during the small cholera epidemic in Summer 1926 at Harbin, some experiments were carried out which may be described here.

In these experiments the leading idea was to imitate natural conditions as far as possible. Almost all of the above mentioned authors had used pure cultures of vibrio cholerae for their investigations. Thus the concentration of the bacilli on the contaminated objects was a far higher one than generally occurring under natural circumstances. For this reason no cultures of bacilli were used in the following investigations but exclusively *fresh stools*, obtained from cholera patients. These excrements were used for contamination of 5-cent-banknotes, which are frequently in circulation and, therefore, very dirty. For that purpose, 20 banknotes were cut into slips and used without previous sterilisation. The method of contamination, referred

later on in detail, tried to imitate likewise natural conditions as far as possible. After contamination the slips were either directly exposed to diffuse daylight, or rolled up in paper.

EXPERIMENT NO. I

A typical rice water stool containing a considerable amount of cholera vibriones was used.

Data about the bacteriological investigation of this stool: Microscopical findings in a mucous flake: Among numerous bacteria of different kinds a small number of vibrio-shaped bacilli are to be seen. Four test tubes, containing peptone water, each inoculated with one loop of stool (mucous flake), showed beginning homogeneous cloudiness already after two hours, becoming more intense during the following two hours. After 3 hours' incubation to one of these tubes one drop of a cholera agglutinating serum (titre 1:3000) was added. Soon typical agglutination with rather big flakes was observed. A microscopical preparation, made from the surface of another of these tubes, showed typical cholera vibriones almost in pure culture. Thus the diagnosis was confirmed four hours after inspection of the stool. After 3, 4, and 6 hours agar plates were inoculated with material from the peptone water tubes.

The results on the following morning were:

1st plate, inoculated after 3 hours: Almost pure culture of vibrio cholerae. Immediate total agglutination of the spirilla by the immune serum 1:300.

2nd plate, inoculated after 4 hours: Practically pure culture of vibrio cholerae. Immediate agglutination with 1:300 immune serum.

3rd plate, inoculated after 6 hours: vibrio cholerae and other contaminating bacteria present.

Methods of contamination:

1st series. The tips of the thumb and the forefinger were dipped for a moment into a flat Petri dish containing the cholera stool. Some seconds afterwards a slip of a banknote was touched with the same fingers, and then laid into a dry, sterile Petri dish. Then the tips of the fingers were dipped once more into the cholera stool and so on. Thus numerous slips were treated and thrown into the Petri dish. The latter was kept in a somewhat darker corner of the laboratory. From time to time a slip was taken out of the Petri dish and thrown into a peptone water tube.

2nd series: The method was almost the same as in I. There was but one difference: A platinum loop was dipped into the

stool, and the material, thus obtained, was ground well between thumb and forefinger, until the fingers became entirely dry. Immediately afterwards a slip of a banknote was touched and so on.

Course of the experiment: After 10, 20, 30, minutes. 1. 2, 3, 4, 5, 6, and 9 hours, slips of series I as well as of series II were thrown into tubes of peptone water. After 3-6 hours, according to the beginning and the intensity of the cloudiness, one or more loopfulls of the material from the upper part of the peptone water were inoculated on Agar-and Dieudonné plates. After 2-3 hours the tubes: series I, 10 min, 20 min, and 30 min, and series II, 10 min show already a slight but typical homogeneous cloudiness. Microscopically agglutinable vibriones could be immediately demonstrated in these tubes.

Findings on the inoculated plates on the next morning:

AFTER 10 MIN.

1st. Series.

50 typ. colonies of vibrio chol.
200 contaminating colonies.

2nd. Series.

Subculture on Dieudonné-plates shows almost exclusively colonies of vibrio chol., giving immediate agglutination with 1:300 immune serum.

AFTER 20 MIN.

Mixed colonies, out of which one third typical colonies of vibrio cholerae. Subcultures show immediate agglutination with imm. ser. 1:300.

50 col. of vibrio chol. 10 colonies of bact. coli. Subcultures show immediate agglutination with imm. serum 1:300.

AFTER 30 MIN.

The Dieudonné-plate shows 20 colonies of vibrio chol. in pure culture; immediately agglutinated by immune serum 1:300.

Colonies of vibrio chol. mixed with colonies of other bacteria; subcultures from vibrio chol. colonies immediately agglutinated by 1:300 immune serum.

AFTER 1 HOUR

Numerous contaminating colonies; after several subcultures v. ch. was obtained in pure culture and identified by agglutination.

No vibr. chol.

AFTER 2 HOURS

Contaminating colonies of different bacteria all about the whole plate.	No vibr. chol.
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AFTER 3 HOURS

Almost pure culture of typical colonies of vibrio chol. showing immediate agglutination with 1:300 immune serum.	Highly contaminated; no vibrio cultures to be found.
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AFTER 4 HOURS

Practically pure culture of typical vibrio cholerae colonies, identified by agglutination.	No vibr. chol.
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AFTER 6 HOURS

No vibr. chol.	No vibr. chol.
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AFTER 9 HOURS

No vibr. chol.	No. vibr. chol.
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The agglutinator power of the strain of vibrio cholerae, cultivated from a slip of a banknote, 4 hours after contamination was compared with that of the original strain. The immune serum used in this case had the titre 1:2000-1:3000.

When the 16 hours old cultures of either of the strains were added to the immune serum, in both cases immediate agglutination was observed in the tubes 1:100-1:300, after one minute in the tubes 1:500, and after 10 minutes in the tubes 1:1000, and after 30 minutes in the tubes 1:2000. On the next morning in the tube: "original strain 1:3000" marked agglutination was observed, while in the tube. "strain from paper money 1:3000" the phenomenon of agglutination was less marked.

Results of the agglutination experiment: The original strain showed perhaps a little higher degree of agglutinability, anyhow, there was only a very slight difference, if at all between the original strain and that cultivated from the contaminated banknote.

EXPERIMENT NO. II.

The same series of experiments were performed as in E. I. but with a cholera stool, *containing only very few cholera vibriones*. The methods applied were the same as in E. I. with but one modification. The contaminated slips were wrapped into a sheet of a newspaper which was exposed to the daylight.

Data about the investigation of the stool: Rice water stool, showing microscopically bacilli, cocci, and spirilli. Tubes of peptone water showed only after 6 hours a beginning cloudiness due to the proliferation of a big bacilliform bacterium. 3 and 4 hours after inoculation agar plates were inoculated with material from the surface of the peptone water. After 20 hours incubation a very small number of typical colonies of vibrio cholerae were found on them, and identified by agglutination, and besides it numerous contaminating colonies. Plates, cultivated 6 hours after contamination showed prevailing colonies of different kinds of bacteria, but no colonies of vibrio cholerae could be cultivated from them.

30 min., 1, 2, 3, 4, 6, and 9 hours after infection two slips from the banknotes, inoculated by the different methods, were taken out of the wrap, and thrown into tubes of peptone water. After 3-4 hours' incubation agar—and Dieudonné plates were inoculated with material from the surface of the tubes.

Results: Vibrio cholerae was alive on the banknotes up to one hour after contamination with the cholera stool (identification of the vibriones by culture and agglutination). After two and more hours no cholera bacilli could be found by the enrichment method.

CONCLUSIONS

1. The cholera vibriones when dried on banknotes, touched by fingers infected with cholera stool, remained alive up to 4 hours.

2. Vibrio cholerae, cultivated from banknotes 4 hours after contamination showed the same cultural and serological qualities as the original strain.

3. During cholera epidemics the use of banknotes is not entirely free from danger.

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PROBLEMS OF CHOLERA IN CHINA AND JAPAN

The importance of the cholera problem in the Orient cannot be over-emphasized. The vast Empire of India is notorious as being the original home of this fatal infection, and scarcely one year passes but thousands of cases are recorded in some part or other of this vast dependency of Great Britain. As we travel eastwards, we find constant outbreaks in Burmah, Siam, French Indo-China, South and Central China, in most of which endemic centres probably exist.

If we take the year 1926 (a rather bad year for cholera) we find:

- a. India, recording at least 72,859 deaths (actual cases probably over 150,000).
- b. Siam, recording 10,655 cases.
- c. French Indo-China, with 19,029 cases (13,925 fatal).
- d. Philippines, with 690 cases (in 1925 there were 1059 cases).

Japan, with her fine quarantine system, was successful in keeping the disease at bay, and reported only 26 cases in that year.

Korea, in spite of a vigorous campaign, had 252 cases with 159 deaths.

South and Central China were badly affected in 1926, serious outbreaks having been reported in almost every city in the Yangtze Valley, such as, Nanking, Soochow, Wusieh, Huchow, Anking, Hankow, Wuchang, etc., besides cities farther south, such as, Foochow, Amoy, Swatow, Canton, Hainan, etc. In Shanghai district, there were at least 20,000 cases, the two Chinese Summer Disease Hospitals admitting 2481 cases in the week, August 1-8.

In Manchuria, which is in close relation with both China Proper and Japan, thanks to close co-operation between the Chinese and Japanese Health Services, only 1500 cases occurred in 1926 as compared with over 10,000 in the previous cholera year of 1919, when no such co-operation existed. It can be definitely said that both in Manchuria and Japan, there are no endemic centres, all outbreaks occurring being traceable to importations from the south.

The task of combating cholera in the Far East is obviously of a two-fold nature.

- (1) Offensive measures to be taken in the endemic centres.
- (2) Defensive measures in areas threatened by importation of the infection.

Solid progress can only be hoped for if the actual fight against the disease in endemic centres is everywhere taken up with equal energy and efficiency. Defensive measures, on the other hand, adopted in cholera-free areas, must be reduced to the absolutely necessary minimum in order not to interfere unduly with commerce and traffic.

Let me concentrate at present upon the defensive measures. The weapons recommended by the International Sanitary Convention of 1926 may first be scrutinised. From the articles dealing with cholera (see Appendix) it may be gathered that the Convention gives vast powers to the sanitary port authorities in the case of *cholera-infected* ships, but limits them in the case of *cholera-suspected* ships and still more in the case of *healthy* ships. In fact, according to Article 33 free pratique should immediately be given to such vessels. Article 34 finally emphasizes anti-cholera vaccination as a specially reliable preventive measure.

The Japanese Delegates at the 1926 Paris Conference, while signing the Convention, reserved the right for their Government "to take all measures considered as necessary by the sanitary authorities against carriers of the *V. cholerae*." Therefore, it is clear that the intention of the Japanese authorities is to continue enforcing rigorous measures for the detection of carriers among ship passengers. The procedure adopted by them may be shortly described as follows:

1. Enforced defecation by all members of crew and passengers on the morning of arrival, and collection of small samples in little Petri dishes.
2. Placing of a small particle of the specimen with sterile toothpick in test tube containing peptone water.
3. Handing over of all the peptone cultures (4-5 hours old) in racks of thirty tubes each to the quarantine authorities.
4. Making of films (five on one slide), drying over a spirit lamp, staining with dilute carbol fuchsin for $\frac{1}{2}$ min., drying between filter paper without washing in water, then examining under the microscope.¹

1. In this way, Dr. Yano of the Naval Quarantine Service could with the aid of another trained doctor assistant inspect six slides, that is 30 specimens, in one minute. While I was in the Laboratory the examination of 280 specimens took just 40 minutes from the time of arrival of the peptone cultures to the final report by telephone to the shipping authorities. In addition to Dr. Yano, there were two medical assistants and six technicians in August 1926 undertaking these fecal examinations for steamers arriving from Shanghai (Wu Lien Teh).

Apparently quite apart from their reservation, the Japanese authorities are technically entitled to enforce these rigorous methods (see end of Art. 29 and also Art. 15 of the Convention²). The fact that stool examinations are not mentioned in the Convention (as contrasted with the recommendation of vaccination) and that the Japanese Delegates reserved the right of their Government to take steps in this direction, however, shows that in other quarters the necessity or perhaps even the value of these stool examinations is doubted. At the F.E.A.T.M. Conference in Calcutta (December, 1927), Colonel Graham, Sanitary Commissioner for India, strongly spoke against the stool examinations as enforced by the Japanese Health authorities. Thus it may be said that there are two schools of thought, one relying mainly upon vaccination, the other regarding stool examinations as indispensable.

Let us try to consider these two views impartially. We all agree that anti-cholera vaccination "is a method of proved efficacy in staying cholera epidemics and consequently in lessening the likelihood of the spread of the disease" (1926 Convention, Art. 34). Another question is whether Vaccination, even when carried out on a large scale, will be instrumental in doing away with carriers of the cholera germ. Opinions on this point are rather divided. Authors like Frankl & Wengraf, Erdheim and Schopper, etc. believe that the number of carriers in an epidemic is diminished by vaccination; Rogers records an instance where "in a camp of some 3,500 Servian prisoners, inoculation during an epidemic produced no bad effect, but on the contrary this measure brought the disease to an end in from 8 to 14 days, while the number of carriers rapidly fell to zero." On the other hand, it is on record that not rarely vaccinated persons become carriers (Kaup, Nomura, Sotoma and Harata). To hope, therefore, that vaccination, even when universal, will abolish the carrier problem, would be vain. Carriers will probably be diminished in number, but at least some will remain.

Turning now to the stool examinations, we must confess that apart from its inconvenience to individuals and the delay

2. Article 15-Any ship whatever its port of departure, may be subjected by the sanitary authority to a medical inspection, and if circumstances require it, to a thorough examination. The sanitary measures and procedure to which a ship may be subjected on arrival shall be determined by the actual condition found to exist on board and the medical history of the voyage.

It rests with each Government, taking into account the information furnished under the provisions of Section I of Chapter I and of Article 14 of this Convention, as well as the obligations it has undertaken under Section II of Chapter I, to determine what procedure is applicable in its own ports to arrivals from any foreign port, and in particular to decide whether, from the point of view of the procedure to be applied, a particular foreign port should be considered as infected.

to traffic it causes, this method is by no means free from sources of error. I am ready to admit that some of these errors may be obviated by the great care and efficiency of the sanitary staff. Such are:

(1) *Prevention of frauds.* Certainly it would be ideal to obtain rectal swabs from each individual, but this would entail so much inconvenience and delay as to be extremely difficult on a large scale. If defecation is left to the individual, great care is necessary to prevent fraud.

(2) *Evaluation of results.* Where a skilful staff is present, there should be little fear of vibrios (if present) being overlooked in the stress of microscopic examination. In this connection, it is more likely that cholera-like vibrios may lead to the detention of harmless passengers.

There is, however, one unavoidable obstacle. As established by many authorities, e.g. by Heiser, Greig, Yagasaki, Noda, Nomura and Harata, carriers may not void cholera vibrios with each stool and not even on each day. In other words, an examination of a single specimen does not entitle us to consider an individual as a non-carrier. Interesting data in this regard were supplied by Amata, Hagawo, Nakamura and Hasegawa among 391 persons quarantined as contacts during the 1922 epidemic at Tokyo, where 33 carriers and 4 cases were found. Of these 37 instances there were detected:

Through one examination	17
„ two „	5
„ three „	12
„ four „	2
„ five „	1
	—	3
		37
		—

Consequently, in order to be sure that no carrier may be missed, it would be necessary to examine travellers not once, but at least two or three times. To detain passengers of a *healthy* ship for such a prolonged period would be absolutely against the spirit of the 1926 Convention.

In conclusion we may say therefore that neither vaccination nor stool examination can be considered as absolutely efficient. Both methods have their weak points as well as their technical difficulties. It would be vain to hope that either will absolutely prevent importation of the disease. Under these circumstances it is clear that defensive measures alone are insufficient to

3. The Japanese references are mainly gathered from "Studies of Cholera in Japan" by Rokuro Takano, Itsuya Ohtsubo and Zenjuro Inouye, Geneva, 1926.

protect a country against the importation of cholera, and one must necessarily pay more serious attention to an active campaign against the disease in neighbouring endemic centres. In the case of China and Japan such measures would comprise:

1) An intensive study of the endemic areas in the Yangtze Valley.

2) Removal of causes wherever possible, e.g. control of water works, sterilisation of wells by chemicals, etc. (A promising beginning in this direction has been made by the Shanghai Chinese Public Health Authorities).

3) Mass vaccination in the endemic areas; vaccination of each passenger leaving for Japan or Manchuria.

4) Fullest cooperation among the two health authorities in the spirit of mutual confidence and helpfulness.

APPENDIX

International Sanitary Convention. 1926.

Cholera.

Article 28.—*Infected Ship*.—A ship shall be regarded as infected if there is a case of cholera on board, or if there has been a case of cholera during the five days previous to the arrival of the ship in port.

Suspected Ship.—A ship shall be regarded as suspected if there has been a case of cholera at the time of departure or during the voyage, but no fresh case in the five days previous to arrival. The ship shall continue to be regarded as suspect until it has been subjected to the measures prescribed by this Convention.

Healthy Ship.—A ship shall be considered healthy, although arriving from an infected port or having on board persons proceeding from an infected area, if there has been no case of cholera either at the time of departure, during the voyage, or on arrival.

Cases presenting the clinical symptoms of cholera, in which no cholera vibrios have been found or in which vibrios not strictly conforming to the character of cholera vibrios have been found, shall be subject to all measures required in the case of cholera.

Germ carriers discovered on the arrival of the ship shall be submitted after disembarkation to all the obligations which may be imposed, in such a case by the laws of the country of arrival, on its own nationals.

Article 30.—*Cholera Infected Ships*.—In the case of cholera, infected ships shall undergo the following measures:—

- (1) Medical inspection,
- (2) The sick shall be immediately disembarked and isolated;
- (3) The crew and passengers may also be disembarked and either be kept under observation or subjected to surveillance during a period not exceeding five days reckoned from the date of arrival.

However, persons who can show that they have been protected against cholera by vaccination effected within the period of the previous six months, excluding the last six days thereof, may be subjected to surveillance, but not to observation.

(4) Bedding which has been used, soiled linen, wearing apparel and other articles, including foodstuffs, which in the opinion of the sanitary authority of the port, have been recently contaminated, shall be disinfected;

(5) The parts of the ship that have been occupied by persons infected with cholera or that the sanitary authority regard as infected, shall be disinfected;

(6) Unloading shall be carried out under the supervision of the sanitary authority, which shall take all measures necessary to prevent the infection of the staff engaged in unloading. The staff shall be subjected to observation or to surveillance which may not exceed five days from the time when they ceased unloading;

(7) When the drinking water stored on board is suspected it shall be emptied out after disinfection and replaced, after disinfection of the reservoirs, by a supply of wholesome drinking water.

(8) The sanitary authority may prohibit the emptying of water ballast in port without previous disinfection if it has been taken in at an infected port.

(9) The emptying or discharge of human dejecta, as well as the waste waters of the ship, into the waters of the port may be forbidden, unless they have been previously disinfected.

Article 31.—*Cholera Suspected Ships*.—In the case of cholera, suspected ships shall undergo the measures prescribed in (1), (4), (5), (8) and (9) of Article 30.

The crew and passengers may be subjected to surveillance during a period which shall not exceed five days, reckoned from the arrival of the ship. It is recommended that the crew be

prevented during the same period from leaving the ship except on duty which must be notified to the sanitary authority.

Article 32.—*Clinical Cholera*.—If the ship has been declared infected or suspected on account only of a case on board presenting the clinical features of cholera, and two bacteriological examinations, made with an interval of not less than 24 hours, between them, have not revealed the presence of cholera or other suspected vibrios, the ship shall be considered healthy.

Article 33.—*Healthy Ships*.—In the case of cholera, healthy ships shall be given free pratique immediately.

The sanitary authority of the port of arrival may prescribe as regards these ships the measures specified in (1), (7), (8) and (9) of Article 30.

The crew and passengers may be subjected to surveillance during a period which shall not exceed five days reckoned from the date of arrival of the ship. The crew may be prevented during the same period from leaving the ship except on duty which must be notified to the sanitary authority.

Article 34.—Since anti-cholera vaccination is a method of proved efficacy in staying cholera epidemics, and consequently in lessening the likelihood of the spread of the disease, sanitary administrations are recommended to employ, in the largest measure possible and as often as practicable, specific vaccination in cholera foyers and to grant certain advantages as regards restrictive measures to persons who elect to be vaccinated.

SCARLET FEVER IN CHINA

CONTENTS

- A. Introduction
- B. Scarlet Fever in Hongkong
- C. Kwangtung—Canton
- D. Chekiang—Ningpo, Hangchow, Wenchow
- E. Kiangsu—Shanghai, Chinkiang, Soochow, Nanking, Hsuehchow
- F. Shantung—Tsingtau, Taikuhsien, Chefoo
- G. Kiangsi—Kiukiang
- H. Hupeh—Hankow, Wuchang, Ichang
- I. Kweichow—Kweichow
- J. Kansu—Lanchowfu, Sining
- K. Chihli—Peking, Tientsin
- L. Manchuria—Dairen, Harbin, Dalainor, Antung, Taheiho
- M. General Conclusion

A. Introduction

It is generally understood that scarlet fever is a disease of temperate climates, being almost unknown in the tropics and seldom seen in subtropical countries. As China (including Manchuria) lies between 20° latitude in the south (Hainan) and 54° latitude in the north (Taheiho, Heilungkiang), great variations may be expected in the incidence of this widespread communicable disease. With a view to ascertaining the exact prevalence of scarlatina throughout the country, a questionnaire was sent to different medical friends, from Canton in the south to Aigun in the north, asking for details regarding the following:

- a. Existence or not, morbidity and mortality.
- b. First appearance in the locality.
- c. Sex and age incidence.
- d. Comparison between Chinese and foreigners.
- e. Any unusual features.

The replies received are not all uniform in character, but enough information is gleaned to enable us to form a fairly accurate idea of its nature in different parts. To supplement these statements, the files of the *China Medical Journal*, customs reports, and all available medical publications were consulted. It will be seen that considerable gaps in the reports exist, but it is hoped that these data may stimulate others to study the question in their respective fields and thus help to explain, among other things, the reason for its unusual virulence in certain regions and even at different times in the same region. Scarlatina is believed to be a recent importation into China and Japan, and its comparative frequency among white people in the south, where few if any Chinese cases are seen, is an interesting phenomenon. W. G. Lennox, in a statistical study of the health of foreign missionary families in China (1,300 marriages and 3,254 Children) came to the following conclusions:

- a. Scarlatina, like other exanthemata, is contracted less frequently inside than outside China.
- b. Out of 193 cases among their children, 104 occurred in North China, 85 in Central China, and 4 in South China. Of these, 12.5 per cent died in North China, 2.3 per cent in Central China, and none in South China.
- c. Altogether 16 deaths from scarlatina occurred among 3,254 children born.
- b. 53 adult missionaries took the disease, making a percentage of 3.4 per cent.

B. Scarlet Fever in Hongkong

Year	Population	Deaths	Cases Plague	Smallpox	Scarlet	Total Infectious Diseases
1904	—	—	510	—	2 (1 E 1 C)	758
1905	—	—	304	—	1	508
1906	326,961	8,379	893	192	1 (C)	1,179
1907	—	7,286	240	341	—	—
1908	336,448	9,271	1,073	472	3	1,668
1911	456,739	7,748	269	272	1	702
1912	467,777	9,682	1,847	709	8	2,757
1913	489,114	8,435	408	111	3	1,013
1914	501,304	9,585	2,146	110	1	2,521
1915	509,160	7,921	144	34	0	507
1916	529,010	10,558	39	712	2 (E)	1,110
1917	—	10,433	38	595	3 (E)	919
1918	561,500	13,714	266	32	73 (2 E 1 C)	1,913
1919	—	11,647	464	27	7 (2 E 5 C)	1,011
1920	648,150	12,419	138	34	3 (2 E)	560
1921	585,880	11,880	150	191	1 (E)	763
1922	662,200	14,569	1,181	212	5 (E)	1,717
		128,591	7,090	3,039	41	15,493

The rarity of scarlet fever in Hongkong can be judged from the above figures. During 12 years (1911-1922), when full records were obtainable, out of 15,493 cases of infectious diseases, only 41 were scarlet fever, and of these, 10 were among Europeans. During the same period, 7,090 cases of bubonic plague and 3,039 of smallpox were encountered.

C. Kwangtung

No municipal records are kept, but individual doctors have written, almost all to the effect that scarlet fever is exceedingly rare among Chinese. For instance:

Dr. W. G. Reynolds and W. W. Cadbury have seen only two uncertain cases (one severe and one mild), both among foreigners, since 1916. The former occurred in a Portuguese boy of eight years, infected from Hongkong.

Dr. P. J. Todd, who has been in practice for twenty-one years in the city, has seen only one suspicious case, but he rather doubts its diagnosis.

Dr. Chas. Selden, who has charge of the Chinese Mental Hospital, accommodating seven hundred patients, has not encountered a single case of scarlatina among its inmates. The only patient he saw was that of a foreigner in Macao in 1898, where the temperature was 102.5° and desquamation lasted for eight weeks.

Dr. S. P. Nye, a Chinese physician who has been in practice for thirty years, has encountered no case.

Dr. Hans Kummels, a German physician with a large practice among wealthy Chinese, reports "a very small number" of cases.

Dr. J. A. Hofmann, for the Hacket Hospital for Women, says no cases have been seen.

Dr. J. M. Margaret, for the staff of Canton Hospital, reports no cases since the last decade.

Dr. Casabianca, in charge of the Doumer (French) Hospital can only recollect one case—that of a Chinese student (native of Kwangsi province) in 1919. "The symptoms were

indubitable and complete with no complications and the evolution was altogether mild." Dr. Casabianca considers the disease quite rare. He has been in practice in Canton for fifteen years.

Dr. E. W. Kir, of the New Zealand Mission Hospital, Kongchuen, Canton, says that since coming to Canton in 1910 he cannot recall having seen any case of scarlet fever in Kwangtung.

D. Chekiang

Ningpo. From the Customs Surgeon's Report, April 1, 1907, to May 31, 1909, we find only one case of scarlet fever, contracted at another port. In 1918-1919 Report, occurs the following:

"A particularly malignant type of scarlet fever rampant in this section. Cases received into the Hospital usually recovered under the serum treatment, but those seen in their homes invariably died."

Hangchow. Report for 1912 mentions foreign cases of scarlet fever with details.

Wenchow. Wenchow is regarded as the dividing line between tropical and temperate China, thus separating a southern non-scarlet from a northern scarlet-producing zone. This arbitrary division is only roughly accurate.

E. Kiangsu

Shanghai. An excellent review is given by Dr. Arthur Stanley (then principal health officer of the Settlement) in his report of 1917, and is herewith reproduced:

"After a period of comparative absence, scarlet fever has become prevalent during the spring of the year, reaching almost epidemic proportions among certain sections of the population. The incidence, compared with what may be termed the initial epidemic of 1902, since when the foreign population has trebled and the Chinese doubled itself, was considerably less, say, about half the incidence of the 1902 outbreak. No special cause can be assigned except the increase of non-immune material. It may be taken, that the majority of persons are to some extent naturally immune to scarlet fever but that during periods of comparative absence of the disease the reservoir of non-immune persons gradually fills up, so that the introduction of a sufficient quantity of infection determines the

onset of an epidemic which lasts until the excess of non-immune persons is removed.

"The first recorded death from scarlet fever in the Foreign Settlement of Shanghai was in 1873. It seems probable that the infection was imported. At about this time, cases are believed to have been reported at Chefoo. The occurrence of scarlet fever in Japan appears to have been officially notified in 1897, but it is probable that a few cases occurred prior to this. Indeed, scarlet fever appears to have reached Japan and China at about the same time and to have been previously unknown.

"By the year 1902 there had been introduced into Shanghai a quantity of infection sufficient to gather epidemic momentum, and the Chinese death record from scarlet fever, in that year 1,500, does not appear to have been exaggerated.

"As would be expected with a recently introduced disease, against which evolution has afforded no natural immunity, scarlet fever has been of virulent type among the Chinese. It is probable that the passage of the disease through the susceptible Chinese has led to an intensification of the virus, so that it is more fatal to foreigners also. The average case-fatality among 68 foreign cases admitted to the Isolation Hospital from 1905 to 1917 was 18.2 per cent. The general case-fatality of scarlet fever in England was in corresponding years below 5 per cent. The tendency in the home countries is for the type to be less virulent, with a case-fatality approximating to 3 per cent. But scarlet fever is characterized by an exceptional variation and in epidemics the case-fatality may vary from 30 per cent to nothing. In Shanghai, there is no indication yet of any general diminution in virulence of type among either foreign or Chinese cases. On the other hand, in Japan the fatality appears to be reverting to the English and American type of low severity.

"There have been in Shanghai curious groups of cases of mild sore throat, without the characteristic signs of scarlet fever, accompanying typical cases. Such mild sore throats appear to have occasionally carried the genuine infection and produced typical cases of scarlet fever. Some of these must be regarded as a typical case of scarlet fever. Others, as cases of tonsillitis, septic, follicular and catarrhal are often accompanied by an evanescent eruption which does not desquamate; and so common as to be likely to be a usual accompaniment of outbreaks of scarlet fever and difficult, sometimes impossible, to distinguish from cases of a typical scarlet

fever. This points to the necessity of isolating all 'sore throats' during an outbreak of scarlet fever of virulent type; but to keep cases not definitely diagnosed as scarlet fever separate from typical cases. As pathogenic organisms may be considered as true to type there seems to be no good reason for thinking that ordinary tonsillitis may develop into scarlet fever, nor that throats susceptible to tonsillitis are *ipso facto* susceptible to scarlet fever.

"In Shanghai there is a tendency towards quinquennial periodicity. The big initial epidemic of 1902 was immediately followed by four years of remarkable absence or reduction of cases; and then a moderate outbreak in 1907, gathering momentum again to maxima in 1912 and 1917. Especially after the initial outbreak in 1902 it would appear as if all the most susceptible material had been exhausted and that a new generation of young children was required before any further great devastation was possible. Above the age of five susceptibility to infection is generally held to become progressively less. As regards seasonal prevalence, the incidence in Shanghai corresponds to the American type, where it is at its maximum in the spring and at its minimum in the fall; whereas, in England, the seasonal variation is just the reverse. The same seasonal variation occurs in Japan as in China. This may be accounted for by the hotter summer in those countries, anything approaching tropical heat appearing to be antagonistic to the propagation of scarlet fever, which is rare anywhere in the tropics and, when introduced, does not seem to be able to retain a hold. In Shanghai, although the maximum incidence is in March, cases may occur throughout the summer; even in July and August with a mean temperature of 80° F.

"Scarlet fever now appears to be pretty generally prevalent in Japan, though the figures are comparatively small. The comparative fatality of the infectious diseases in Japan roughly have the following order in general mortality figures during recent years: typhoid fever, dysentery, cholera, smallpox, plague, and then scarlet fever. Scarlet fever also occurs in the larger towns of Korea.

"In China, scarlet fever seems to be generally prevalent in the Yangtze valley, at least as far up as Hankow; also in the north, in Chefoo, Tientsin, Peking, and Manchuria. In Hongkong, four cases of scarlet fever were reported to have been brought in by two British warships from England in 1898 and that the disease had previously been 'practically unknown' in the Colony. From 1908 to 1916 fifteen cases were notified. In Singapore, Penang, and the Philippines, scarlet fever does not appear to come into the picture so far as health statistics show."

INCIDENCE OF SCARLET FEVER REVISED TO 1922 Foreign Settlement of Shanghai

	FOREIGN CASES NOTIFIED	ISOLATION HOSPITAL						TOTAL DEATHS		
		Foreign			Chinese			Case Fatality	Foreign	Chinese
		Cases	Deaths	Case Fatality	Cases	Deaths				
1873										
1882										
1888										
1889										
1893										
1897										
1898	4									
1899	8									
1900	7									
1901	15									
1902	58									
1903	101									
1904	6									
1905	15									
1906	5									
1907	10									
1908	58									
1909	25									
1910	9									
1911	32									
1912	15									
1913	49									
1914	32									
1915	24									
1916	15									
1917	27									
1918	113									
1919	33									
1920	19									
1921	29									
1922	28									
	27									
	764									
		761	118	15.5	1,071	270	25.2	179		3,782

*F. and C. 160 cases (28 deaths) 17.5%.

Notes on the Table

1. Incomplete notification reduces the value of the first column.
2. Decimal points omitted from case-fatality percentages.
3. Total foreign death figures in Shanghai may be regarded as the most accurate of the data available.
4. The higher case-fatality among Chinese cases in Isolation Hospital does not necessarily indicate a higher virulence of type or lessened resistance among the Chinese as compared with foreigners, for many Chinese cases are brought in moribund after outside treatment had failed.
5. The discrepancy between the foreign deaths in Isolation Hospital and the total deaths is accounted for by cases from outside the settlement being also admitted.

(Foreign cases prior to 1905 isolated in General Hospital.)

Prior to 1902 death figures not regarded as sufficiently accurate to quote; but it may be assumed that scarlet fever was either entirely absent or sporadic in its incidence.

Chinkiang. For the six months ending September 30, 1910, we read: "Scarlet fever during the whole year. Attended four cases, notified six. Sporadic cases occurring in summer is unusual, writer laid the blame on the refugee camps, where he actually found scarlatina."

The reports of April to September, 1914, state: "Scarlet fever and diphtheria have again occurred freely among Chinese, but I have not met with foreign cases." In the next year: "Scarlet fever has been somewhat prevalent, and one case (slight), which was however followed by very profuse desquamation, occurred in the Concession."

In 1915-1916: "Scarlet fever has not been in evidence above the average."

In 1918: "Several cases were seen among Chinese, none among foreigners."

Soochow. The reports state that in 1916 the usual number of scarlet fever cases were seen among Chinese. In 1918, the disease appeared to have been endemic during the whole year.

Nanking. The University Report states that in 1917 scarlet fever was very prevalent in the spring and winter months.

Hsuchow. In spring, 1918, scarlet fever was very prevalent in a severe form, sweeping whole towns and hamlets.

F. Shantung

Tsingtau. We are unable to procure the reports of the Health Department, which will be surely interesting.

Taikuhsien. In 1920, thousands were attacked by scarlet fever and diphtheria.

Chefoo. Hagg relates a mild epidemic in the school of the China Inland Mission for foreign children, evidently imported by steamer. No deaths resulted. (*C. M. J.*, 1918, p. 239.)

G. Kiangsi

Kiukiang. In 1912, two cases were recorded as having been imported from Shanghai.

H. Hupeh

Hankow. Dr. Robert Aird has kindly supplied the following information regarding this district, and refers us particularly to a considerable outbreak in 1917 mentioned in the Consular Medical Report for the year 1916-1917:

"Some anxiety was caused by the undoubted presence of scarlet fever among the Chinese population during the spring months (1917), and steps were taken by the Municipality, by means of leaflets in English and Chinese, to bring home to residents the need for care in order to prevent the spread of infection. The actual extent of the prevalence of the infection in the native city and in the neighboring cities of Wuchang and Hanyang was difficult to gauge, as cases of diphtheria and of simple angina were also occurring about the same time. That the scarlet fever was of a malignant type was shown by the cases actually met with. Thus a Chinese mother and child were brought into the Roman Catholic Mission Hospital one evening, with bad throats and severe rash, and both died within twenty-four hours. A house coolie living in the British Municipal buildings was taken ill and died within four days. A compradore's child in the German Concession was taken ill and recovered, but the compradore caught the infection, and died after a very few days' illness. From a patient in the London Mission Hospital the English nurse in charge unfortunately contracted the infection and developed a very severe attack, and died in five days. The presence of this deadly disease in the midst of a great Chinese population, new to it and without any acquired immunity to it, constitutes a very grave problem, especially in view of the long period during which the disease is contagious, the overcrowding of Chinese dwellings, and the impossibility of adequate isolation and disinfection. It emphasizes the undesirability of crowding Chinese tenement dwellings within the narrow confines of the Concession, for if the infection became at all prevalent in them the foreign population would be almost certain to suffer severely also, especially the children, who are more susceptible than adults."

I have been in practice in Hankow since March, 1904, and the epidemic above mentioned was certainly the most severe we have had during these twenty years, but sporadic cases turn up nearly

every winter. We do not have many cases among Chinese, but those seen in foreigners can generally be traced to a Chinese source of infection; e.g., a Russian child in a good home contracted the disease about a year ago, and it was subsequently found that a jobbing tailor who had been working in the house, had had his own child ill with the disease at home.

1. Whether the above epidemic was the first appearance of scarlet fever in Hankow or not, I am not quite certain, but I rather think it was not. I remember before that time that a British child contracted the disease a couple of days after its father returned from a visit to Shanghai, but there were no subsequent cases of infection from this child.
2. I am sorry I have no tables of scarlet fever, as compared with other communicable diseases. Hankow has so many different authorities in its various concessions, and native city, that there is no central organization for the collection of such data.
3. Comparison between foreigners and Chinese is difficult, owing to the absence of statistics, but personally I have seen more cases among foreigners than among Chinese.
4. Severity among Chinese and foreigners. Those cases which I have seen among Chinese were more severe than those among foreigners, but I have not seen enough to be able to express much of an opinion on this point.
5. Scarlet fever increasing or decreasing. I should say, neither. It occurs irregularly during the colder months.
6. Case charts. I am sorry I have none on hand. My partner, Dr. Skinner, tells me that a case he had last spring in a young English woman showed a typical chart. The Russian child whom I attended last winter had also a characteristic temperature curve, somewhat prolonged by a persistent streptococcal infection of the nose.

Wuchang. Scarlet fever was rampant in this center in 1918.

Ichang. In the Report for 1916-1917, it is recorded that "scarlet fever which was so prevalent in the lower river ports did not reach Ichang."

I. Kweichow

Kweichow. A few cases were reported here in 1918.

J. Kansu

Lanchowfu. In 1919: "There are epidemics occasionally ofscarlet fever. It is surprising that it should be regarded as a disease newly imported to China. I have seen it both in Chinese and foreigners in far inland provinces. Sometimes we seem to have it in very malignant forms." (King, *C. M. J.*, 1919, p. 38.)

Sining. This fever never leaves us. Every autumn and winter finds some children suffering, but every few years it ap-

pears among us in a more malignant form and cuts off children in large numbers. The malignant form has been prevalent this winter and hundreds of children have died. In one village of forty families, sixty children died. (*C. M. J.*, 1916, p. 392.)

K. Chihli

Peking. The Government Infectious Diseases Hospital was established in 1915 in the northern section of the capital. We have not been able to procure any published statistics, but Dr. Yen Chi-chung, the director, has been kind enough to supply the following private information in form of a table:

Year	Sex		Total	Cured	% Cured	Death	% Death	Left Before Time
1915	7M	8F	15	6	40.0	9	60.0	0
1916	110M	94F	204	152	75.0	35	17.0	17
1917	38M	30F	68	46	68.0	17	25.0	5
1918	4M	4F	8	6	75.0	1	12.50	1
1919	4M	0F	4	4	100.0	0	0	0
1920	8M	6F	14	12	86.0	1	7.0	1
1921	41M	41F	82	59	72.0	19	22.0	4
1922	89M	59F	148	109	73.0	33	23.0	6
1923	51M	44F	95	77	81.0	18	18.9	0
	352M	286F	638	471	73.8	133	20.8	5.3%

Private practitioners also report a large number of cases every year, sometimes mild, but often fatal. The latter are usually of the septic type, showing marked vomiting, high fever, headache, red tongue, swollen throat, etc. Complications, like adenitis, otitis, nephritis, rhinitis, are quite common, but rheumatism, myocarditis, or endocarditis is rarely encountered. Scarlet fever, because of its newness and withal virulence among the community, is much feared by the well-to-do classes, but so far insufficient educational work has been done in the way of communal hygiene, and the disease has lately been allowed to affect all classes.

Tientsin. For nearly fourteen years, we have observed very severe cases in this city among both wealthy and poor people. Owing to the widespread ignorance and superstition of the women folk, even after having undergone a general education, infection generally spreads from child to child in the same family until almost every one is attacked. The constant communication between the children and servants of the locality also helps to spread the disease broadcast. Even adults are often attacked, as the following cases show:

- a. An experienced Chinese old-style physician, aged sixty-two (whose son is one of our medical officers), attended

in February, 1923, the family of the late General Chang Hsun for scarlet fever. He himself developed high fever, sore throat, headache, swollen glands, and later nephritis, etc. Within thirty-six hours, he was speechless and delirious, but with stimulants and serum he gradually recovered after four months. A younger son, seeing the serious condition of the father, remained in the same room for most of the time and even slept in the same bed. Three days after he showed all signs of a severe infection with vomiting, pneumonia, etc., and died the same evening.

A married daughter (aet. 28), who had been nursing the father, also became ill and returned to her house with shivering, headache, sore throat, dry cough, and severe vomiting. Her temperature rose to 106°, with pulse of 126, when the rash appeared. Serum was given and after three days the disease abated and she slowly convalesced. Her three children, though isolated early, caught the infection, resulting in the death of the youngest.

- b. Among the Chang Hsun family, which caused the above cases, one adult lady and two children died, though several persons were sick.

These rapid passages were duplicated throughout the Tientsin district during the winter of January to February, 1923, and should give a serious warning to those in charge of public health work regarding the need of establishing more hospitals for the proper reception and care of such cases.

L. Manchuria

From Manchuria, we have fuller statistics. The South Manchurian Railway has a well trained staff all along the line, where well equipped hospitals are built. Their reports, therefore, provide fruitful study. It is ascertained that scarlet fever was first seen in 1908. In 1909, two cases appeared in the Dairen area, then a few more along the line. Since then, sporadic cases have been frequent, resulting now and then in small epidemics. From 1915 till February, 1924, Dr. Tsurumi was the chief medical officer of the Sanitary Department, and to him we are obliged for the following information regarding conditions along the South Manchurian Railway.

In a report published in the *Japanese Journal of Therapeutics* (February 11, 1922) Tsurumi dealt with two epidemics seen by him, namely January to July, 1916, when 118 cases occurred, and January to July, 1920, when 138 cases occurred. The accompanying two tables are interesting:

Dairen Cases (1911-1919)*

1911	101	cases with	22	deaths	21.7%
1912	158	„ „	4	„	2.5%
1913	134	„ „	32	„	23.8%
1914	182	„ „	32	„	17.6%
1915	145	„ „	15	„	10.3%
1916	227	„ „	23	„	10.0%
1917	135	„ „	16	„	11.8%
1918	79	„ „	2	„	2.5%
1919	88	„ „	7	„	7.9%
	<hr/>		<hr/>		<hr/>
	1,249		153		12.2%

	1916		1920
Albuminuria	36, i. e., 25%	Albuminuria	18, i. e., 13.1%
Lymphadenit cerv. .	35, i. e., 24%	Lymphadenit cerv.	47, i. e., 34.3%
Otitis media	21, i. e., 14.7%	Otitis media	10, i. e., 7.3%
Bronchitis	11, i. e., 7.7%	Bronchitis	23, i. e., 16.8%
Enteritis	8, i. e., 5.6%	Nephritis	15, i. e., 10.9%
Nephritis	2, i. e., 1.4%	Uraemia	1, i. e., 0.7%
Rhinitis	2, i. e., 1.4%	Haematuria	7, i. e., 5.1%
Urticaria	2, i. e., 1.4%	Others	16, i. e., 11.7%
Uraemia	1, i. e., 0.7%		

His conclusions are thus summarized:

1. Out of 143 cases personally seen by him, all but three had tonsillitis and angina.
2. The symptoms were severe in 88, mild in 55. Enlarged tonsils predisposes to severe form.
3. Vomiting and diarrhea were seen in the early stages in one third of the cases.
4. Lymphadenitis was frequent in 1920 epidemic. Out of 16 pus examinations, streptococci were seen in 13 (81.2 per cent).
5. Antistreptococcic serum produced good results. Out of 16 cases treated, 4 had immediate benefit (25 per cent), 6 had fair benefit (37.5), while the remainder showed little reaction.
6. Serovaccine was found satisfactory, especially in cases with severe angina.
7. Among 99 admissions into Dairen Hospital in 1920, 15 (15 per cent) showed kidney complications. For three cases, saline infusion combined with sodium carbonate gave excellent results.

Harbin. The following figures are kindly supplied by Dr. Shapiro, in charge of the Harbin Town Infectious Diseases Hospital (mainly for Rusians).

*None of the cases older than ten years. September, 1914, to August, 1915, 163 cases admitted in hospital. More than half under five years, others six to ten. More female than male. This corresponds to 1914 London statistics.

Year	Total Admission Infectious Diseases	Scarlet Fever	Deaths
		Cases	
1920	377	22	1 (R)
1921	496	36	4 (R)
1922	739	63	5 (3R 2C)

The only two Chinese admitted died; evidently they went to hospital only after all hope had been given up at home.

A localized outbreak occurred in 1923-1924 in the Refugee Children's School organized by Madam d'Anjou (wife of the commissioner of customs). A short interval existed between two groups of cases; namely, the first, lasting from November 11 till November 17, 1923, during which four out of thirty-six children were attacked; the second, from January 5 till January 19, 1924, when thirteen were attacked. The epidemic was mild and no deaths resulted. Several teachers and attendants complained of sore throat, but did not show any actual disease.

Our medical officers saw a considerable number of severe cases among Chinese in 1923. Among these, was the family of a postal clerk (Fukien) who lost all his three children (aged seven, five, two) within the short space of four days (November 25-28). Two cases were admitted into our hospital on December 8, 1923; received serum treatment; both recovered. These were apparently infected by neighboring mild cases.

The son (aet. 8.) of a prominent French resident suffered from a mild attack of scarlatina in 1922. Severe nephritis and heart weakness later appeared and kept on for months, but patient recovered.

Dalainor. In October, 1923, some excitement was caused by the sudden outbreak of scarlatina in the coal-mining village of Dalainor, where pneumonic plague raged violently in 1921. Our medical officer, Li An, investigated this matter and reported as follows:

Scarlet Fever in Dalainor

1923-1924, Chinese 1 case (no death)
Russian 50 cases (5 deaths, all male).

—
Total 51 cases

Sex:	35 male, 16 female			
Age	1 year	3	11 years	2
	2 years	4	12 "	0
	3 "	5	13 "	1
	4 "	5	14 "	1
	5 "	9	15 "	2
	6 "	1	22 "	1
	7 "	3	30 "	1
	8 "	5	35 "	1
	9 "	5		
	10 "	2		

Incidence month by month:

October, 1923 (19th)	1 case
November	9 cases
December	7 „
January, 1924	30 „
February	4 „ (last case, February 19.)

Evidently the epidemic was not very severe, both among Chinese and Russians.

Antung. Scarlet fever was noticed almost every year, usually in a severe form. The "Antung Medical Report" for 1921 states: "In the autumn a severe epidemic occurred with severe mortality among children. In one family, all the children died within a few days."

Newchwang. Dr. Phillips attended several foreign cases in 1918; none died. At the same time numerous cases with a large percentage of deaths occurred among Chinese. No severe epidemic since then.

Taheiho. This northern town of Manchuria was invaded by scarlatina for the first time in October, 1923. Our medical officer, W. H. Shih, provides the following report:

"During the months of October and November, I saw twenty-two cases altogether, including both hospital and outside. All of them were children ranging from two to ten years of age. Among the twenty-two cases there were five deaths and the other seventeen cases ran a comparatively normal course. The following description roughly represents most of the non-fatal cases:

"The onset is sudden, with vomiting, headache, and fever, followed later on by sore throat. On the following day, the rash appears first on the chest and abdomen and later on extends to the limbs and buttock. The face is flushed and eyes red. Pulse fast and full, fever goes up to 104° or 105°. Urine scanty and cloudy. Bowels constipated. This generally continues till the end of five or six days, when the fever subsides and gradually returns to normal with desquamation.

"In one malignant case, a schoolgirl of eight years, I noticed that the onset was nearly the same as the others, but more severe. On the following morning, the rash came out abundantly all over the chest and abdomen, also around the buttock. Within a few hours, the papules were full of pus, so that to the naked eye they were white elevations instead of red. In the afternoon the patient was delirious and died on the third day.

"As I was unable to obtain either antitoxic serum or vaccine in Taheiho and Blagoveschensk, I treated the first few cases with general principles; i.e., well ventilated room, rest in bed, light diet, and painting of tonsils occasionally with formalin (1 in 100) and lastly isolation.

"Later on, through the kindness of Dr. Lin C. S., I was able

to obtain some anti-scarlatina vaccine from Harbin. I tried it on four cases, both recovered; also gave prophylactic injections to four children.

"Of the five fatal cases, two were girls about eight years old, who died on the third and fifth day, respectively. The other three cases were a boy of ten and two girls of two years old; all died of throat complications following suppuration of cervical glands."

M. General Conclusions

1. Scarlatina is practically absent or very mild in South China, not unduly severe in Shanghai and central provinces, and severe in the north.

2. The disease seems to be comparatively more frequent among Westerners residing in the country. In the north, although several deaths have been recorded among them, infection is usually not so severe as among the same classes of Chinese. The statistics on missionary families, compiled by Dr. Lennox and briefly referred to elsewhere in this article, form instructive reading.

3. The special virulence of the disease manifested when Chinese are attacked in epidemic form may be explained partly by its recent introduction into the country before any natural immunity is yet evolved. But the example of Japan, which, owing to her complete hospitals, and thorough system of notification and isolation, has only allowed a mild and infrequent form to appear among the people, is sufficient to teach the responsible authorities in China the best and quickest way of subduing the disease.

4. Where septic conditions exist, treatment with a combination of antistreptococcic serum and diphtheria antitoxin often produces satisfactory results. But in view of the rapid progress of the disease in individual patients, this method must be applied at the earliest possible opportunity.

5. From recent investigations upon the bacteriology of scarlet fever, especially by Drs. George and Gladys Dick, it seems that two substances, of the nature of toxins, play an essential part in the morbid phenomena of the disease, and that these toxins may be neutralized by antitoxins, thus bringing us nearer to a condition familiar in diphtheria. Further, the injection of scarlet fever toxin may provide a method for distinguishing between susceptible and non-susceptible individuals, in the same way that the Schick test has done in diphtheria.

6. With the immense amount of material at hand in North China, especially Peking and Tientsin, it is hoped that much intensive research work may be carried out by trained men, of whom there are not a few now. Only by such means can the peculiar conditions in this region be satisfactorily studied with benefit to both foreigners and Chinese.

THE SCARLET FEVER PROBLEM IN THE FAR EAST

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Although scarlet fever is mild and rarely seen in the Southern provinces of China, it causes severe widespread epidemics in the North. The paper by Yang Ting Kuang and W. H. Shih²¹⁾ shows that scarlet fever is very rare in Hong-kong. (During years 1912-1922, out of 15,492 cases of infectious diseases only 41 cases of scarlet fever were recorded). Some of these few cases might have been imported, as this disease is practically unknown among the native population of Kwangtung.

In the Central Provinces, with temperate climate, scarlatina is more frequent during the cold season. Here numerous outbreaks often show a high mortality. For instance, from 1905 to 1917 the mortality among foreign patients at the Shanghai Isolation Hospital amounted to 18.5% (583 cases, 118 deaths). The 1924 Annual Report of the Shanghai Public Health Department shows an increase in the incidence of scarlet fever. Of 45 Chinese cases admitted to the Chinese Isolation Hospital, there were 8 deaths (17.8%). According to this report, scarlet fever in Shanghai is often of an intensively virulent nature, the mortality ranging from 8-25%.

In the spring of 1917 a severe epidemic of malignant scarlet fever arose in Hankow (Hupeh) with numerous fatal cases among the native as well as foreign population. Severe epidemics were also reported from Sining (Kansu) breaking out usually in autumn and winter. The 1916 epidemic there, for instance, killed hundreds of children⁶⁾.

The province of Chihli is likewise severely affected by scarlet fever. The statistics of the Hospital for Infectious Diseases (Peking) show a mortality of 18.9% in 1923. The same applies to Tientsin. In 1916 and 1920, larger epidemics occurred in the area of the South Manchurian Railway with a relatively high mortality: Tsurumi¹⁹⁾ reported an average mortality of 12.2% from 1911-19 (1,249 cases with 153 deaths).

In Harbin, as elsewhere in North Manchuria, its severity varies. While it may be quite benign sometimes among Russians, on other occasions it claims an exceedingly high mortality. The Reports of the Harbin Town Infectious Disease Hospital, kindly supplied by Dr. Bergmann, demonstrate this fact in an effective manner:

TABLE I.

Harbin Municipal Hospital. Summary of Scarlet Fever Patients Treated.

No. of patients:									Died:								Mortality %
Year	Adults m.	Adults f.	Children	Russians	Chinese	Jews	Other nat.	Total	Adults m.	Adults f.	Children	Russians	Chinese	Jews	Other nat.	Total	
1918	—	—	20	7	1	12	—	20	—	—	2	1	—	1	—	2	10.0%
1919	—	1	17	11	—	4	3	18	—	—	—	—	—	—	—	—	0.0%
1920	2	5	15	15	—	5	2	22	—	—	1	1	—	—	—	1	4.55%
1921	1	5	30	20	2	14	—	36	1	1	2	2	2	—	—	4	11.11%
1922	5	3	51	42	2	10	5	59	—	—	6	3	2	1	—	6	10.19%
1923	2	4	98	66	—	34	4	104	1	—	16	15	—	2	—	17	16.35%
1924	6	7	103	90	2	16	7	116	1	—	15	14	1	1	—	16	13.79%
1925	5	3	106	80	8	16	10	114	1	1	7	3	4	—	1	8	7.01%
I-Vth month																	
Total	21	28	440	331	15	111	31	489	4	2	49	39	9	5	1	54	11.04%

The high morbidity of scarlet fever among the Russian population in the Municipal area of Harbin (average population 50,000-86,000) is shown as follows:

TABLE II

Incidence of Scarlet Fever at Harbin (Pristan and Novy Gorod) 1909-1924

Year	Cases	Year	Cases	Year	Cases	Year	Cases
1909	201	1913	313	1917	119	1921	202
1910	269	1914	321	1918*	51	1922	149
1911	337	1915	152	1919*	75	1923	270
1912	222	1916	116	1920*	103	1924	218

Average number per 1,000 of population for 16 years—2.85
* Incomplete.

Among the Chinese population in Harbin the attacks of scarlet fever have usually been of a severe character, as has been personally observed by us.

Generally speaking, scarlet fever is more severe in North China than in corresponding parts of Europe and America. For instance, the Austrian Public Health Reports show that the total number of scarlatina cases from April 12th to May 16th, 1925 were 752, of which 3 died (0.3%).

Also in Western Europe, North America and Australia, scarlet fever appears in a comparatively mild form as may be seen from the available data drawn from the Annual Report, 1924, of the League of Nations:

England 1923	cases	85,603	
				deaths	993	=1.17%
Canada 1924	cases	17,340	
				deaths	393	=2.26%
Australia 1923	cases	6,634	
				deaths	45	=0.69%
New Zealand 1924	cases	1,167	
				deaths	13	=1.11%

On the other hand, in Japan, though scarlet fever is not so very prevalent, the type is also more severe than in Western Europe as may be seen from the figures:

Incidence of Scarlet Fever in Japan, 1919-1924¹⁴⁾.

Year	Cases	Deaths	Fatality rate
1919	1,325	109	8.2%
1920	1,368	90	6.6%
1921	1,589	82	5.2%
1922	1,657	97	5.2%
1923	1,562	88	5.6%
1924	1,843	121	6.6%

Korea Statistics:

Year	Cases	Deaths	Fatality rate
1920	371	106	28.6%
1921	717	209	29.1%
1922	585	239	40.4%
1923	1,008	140	25.0%
1924	1,361	338	24.8%

Such high morbidity and mortality in the Far East call for the need of fighting this infection with all weapons provided by modern science.

Investigations on scarlet fever have produced important results during the last few years. Tests have been introduced which enable us to determine the individual susceptibility to scarlatina, (Dicks' skin test). Other tests (Schultz-Charlton blanching phenomenon, Pastia's sign) have been introduced for diagnosis. In America special emphasis has been laid upon the important rôle played by *Streptococcus haemolyticus* in the causation of this disease. On the basis of these investigations, and partly independently of them, a new therapeutic scheme has been worked out.

Owing to the important results which may be expected, our Chief, Dr. Wu Lien-Teh, has suggested a thorough study in Harbin and Manchuria of this problem.

Our investigations are not yet completed, and only a preliminary summary is therefore given here.

The frequency of *Streptococcus haemolyticus* in scarlet fever patients was noted already in 1885 (Crooce). According to Jochmann¹²⁾ in post mortem 70% *St. haem.* was found in the organs of scarlet fever corpses. Since 20 years ago, when the use of Schottmuller's blood agar plates was adopted, fuller knowledge about the frequency and biological features of *St. haem.* in the throat and tonsils of scarlet fever patients was possible.

Gordon¹¹⁾ found in nine out of ten tonsillar swabs abundant colonies of *St. haem.* (90%). In four of these *Streptococcus haemolyticus* exceeded all other bacteria grown on the plate.

Dochez and Sherman⁹⁾ say that during a certain period of the disease *St. haem.* is to be found in every case and shows distinct biological features. Bliss and Bristol⁵⁾ found *St. haem.* in 100% (often in pure cultures) in swabs from throats of scarlet fever patients.

Our own investigations so far carried out in Harbin tend to confirm the above statements, as may be seen from the figures supplied later on.

Cotton swabs were prepared for taking the material from the pharynx without touching the tongue or other parts of the mouth, and examined within three hours. In every case rabbit blood agar recently poured into plates was used. (1 cc. of defibrinated blood mixed with 5-6 cc. of agar). The throat

material was then streaked over the surface of the first plate. 5 more plates were inoculated in succession with a sterile bent glass rod which had been rubbed over the surface of the first plate.

TABLE III.

No.	Race.	Age.	Sex.	Res.	Date material taken	Days sick	Growth of <i>St. haem.</i>	Practically pure cult. on plates:	REMARKS.
1	Ch.	6	m.	d.	19. VI	4	pos.	3. 4. 5. 6	O. P. Parents refused to keep him at hosp.; died at home.
2	R.	8	m.	r.	21. VI	5	pos.	4. 5. 6	
3	R.	3	m.	r.	23. VI	5	pos.	(1. 2) 3. 4. 6	
4	Ch.	22	f.	d.	27. VI	3	pos.	4. 5. 6	
5	Ch.	8	f.	d.	27. VI	4			
a)	sl.	fr.	nose				pos.	5. 6	Relatives of case 1; stay hospital
b)	sl.	fr.	throat				pos.	4. 5. 6	refused; died at home.
6	R.	11	f.	d.	8. VII	6	pos.	mixed	Swabs taken 12 h. before death: thick membr. on throat.
7	R.	4	f.	r.	8. VII		pos.	3. 4. 5. 6	Mild.
8	R.	8	f.	cr.	9. VII	11	pos.	2. 3. 4. 5. 6	Mild myocarditis; recovered quickly
9	R.	10	m.	c.					
a)	first swab				22. VII	9	pos.	4.	Adenitis, pyemia, thick membr. on throat.
b)	second swab				5. VIII	23	pos.	contam.	
10	R.	6	f.	cr.	27. VII	4	pos.	2. 3. 4. 5. 6	Adenitis.
11	R.	10	f.	r.	30. VII	5	pos.	2. 3. 4. 5. 6	Mild, without comp.
12	R.	6	m.	r.	4. VIII	5	pos.	3. 4. 5. 6	
13	R.	4	f.	cr.	10. VIII	7	pos.	mixed	Plenty memb., severe case, mixed with diph.-like bac.
14	R.	4	m.	r.	10. VIII	6	pos.	3. 4. 5. 6	
15	R.	1½	m.	d.					
	1st investigation				12. VIII	4	pos.	(3) 4. 5. 6	Died on 12th day of disease.
	2nd investigation				18. VIII	10	pos.	5.	
16	R.	2¾	f.	r.	19. VIII	15	pos.	2. 3. 4. 5. 6	
17	R.	3½	m.	r.	19. VIII	8	pos.	2. 3. 4. 5. 6	Mild.
18	R.	9	m.	r.	7. IX	7	pos.	contam.	Plates contam. with num. small colonies of small streptobac.

d=died.
r=recovered.

c=complication.
R=Russian.

Ch=Chinese.
O. P.=outpatient.

These figures show that in every one of our cases (21 examinations) *St. haem.* was present in the throat (100%). In 17 of these, streptococci were found in practically pure culture on the last plates of the set (81%). It is significant that in

cases with thick membranes more contaminations and mixed infection were seen than in the mild cases with no membranes. The purest cultures were as a rule obtained in the early stages of the disease (4-5th day). Sometimes in later stages of mild cases (8-15th day, see cases Nos. 16, 17) practically pure cultures of *St. haem.* were obtained from the tonsils.

Stevens and Dochez¹⁸⁾, Bliss⁴⁾, Tunicliff²⁰⁾, Gordon¹¹⁾, etc., emphasize that the streptococcus of scarlet fever serologically represents a specific type which is differentiated mainly by agglutination and absorption of agglutinin test from other streptococci.

Morgenroth, Schnitzer and Munter found that in mice, when *St. haem.* changes into a green producing type, there is considerable loss of virulence.

If we could elaborate a method by which such a change could be quickly produced in the human organism, an excellent remedy against scarlet fever would then be available.

G. and G. Dick were able to produce scarlet fever-like phenomena in human beings by planting upon the naso-pharyngeal mucous membrane of several volunteers a culture of *St. haem.*, obtained from scarlet fever patients. Thus all Koch's postulates seem to have been fulfilled by them.

These authors also made numerous studies on the toxin of these streptococci. They state that the toxin has certain features and when filtrated can be applied as follows (Zingher²²⁾):

- 1) Determination of the susceptibility and immunity to scarlet fever.

- 2) Active immunization with sc. f. toxin and determination of its effect.

- 3) Help in the diagnosis of doubtful cases of scarlet fever.

- 4) Studies on the nature of scarlet fever toxin and antitoxin.

- 5) Standardizing the antibody content of antitoxic serum.

- 6) Identification of *St. haem.*, from doubtful cases of scarlet fever.

- 7) Epidemiologic studies: Determination of the presence of specific *St. haem.* in normal throats (carriers) and in patients with scarlatina sine exanthemate.

- 8) Studies on the germs of postscarlatinal infection.

Out of these applications of Dick's toxin, the first is of great importance. If the susceptibility to scarlatina is determined by systematic investigations, susceptible persons, especially in schools, institutions and communities might be immunized. This would help to prevent the distribution of this disease as in the case of diphtheria. Among the lower classes of the Chinese population, where poverty and lack of personal hygiene make other measures difficult, the prophylactic treatment with antiscarlet fever toxin would be of great use.

For this purpose we have undertaken a large number (nearly 2000) of skin tests among the Chinese of Fuchiatien (Harbin).

At first the original standard toxin which Drs. Dick and Dick kindly presented Dr. Wu Lien-Teh at Chicago was employed. Later on, our own toxin obtained from strain of case 15 (Table III) was used. The strength of this had been compared with the standard toxin on the patients of our hospital at Harbin. For skin tests we dilute the toxin 1:1250.

Our studies in this direction are still being pursued, and only a few of the results obtained are given here.

The intradermal tests were made exclusively on the upper arm and were read off within 20-24 hours. They are marked as —, +, ++,

+++, just as the Dicks recommend.

+ 1.5 cm. diam. or under.

++ 1.5 to 3 cm.

+++ over 3 cm.

TABLE IV.

Dick Test with Different Age Groups (Chinese in Harbin).

Age	Dick neg.	Dick positive				Total	Per Cent Dick posit.
		one	two	three	plus.		
1-4 1	1	2	11		15	93.3%
5-9 11	10	9	6		36	69.4%
10-14 95	46	34	19		194	51.0%
15-19 146	113	38	6		303	51.8%
20-24 114	72	36	2		224	49.1%
25-29 87	59	14	—		160	45.6%
30-above	. 214	94	34	1		343	37.5%

The above shows a high percentage of positive reactions as compared with those made in other countries. We use Zingher's table:

TABLE V.
New York City, William Parker Hospital.

Age	Total tested	Dick pos.	Dick neg.	% Dick pos.
0-6 months	29	13	16	44.8%
6-12 „	52	34	16	65.3%
1-2 years	233	167	66	71.6%
2-3 „	204	131	73	64.2%
3-4 „	241	146	95	60.5%
4-5 „	264	128	136	48.2%
5-10 „	1955	678	1277	33.6%
10-15 „	2965	677	2288	22.8%
15-20 „	981	166	815	16.8%
20 years up	776	112	662	14.4%
Total	7700	2252	5448	29.2%

TABLE VI. Rosen and Corobicina.
Children of the Closed Infants' Home in Moscow. 1187 cases.

Age	No. tested	% Dick pos.	Age	No. tested	% Dick pos.
0-1 year	68	47.0	7-8 years	203	53.2
1-2 years	38	68.8	9-10 „	289	43.2
2-3 „	43	65.1	11-14 „	339	38.3
3-4 „	24	83.3	15-16 „	35	48.5
4-6 „	100	67.0	19 years up	48	35.4

Our high percentage of positive reaction demonstrates clearly that scarlet fever encounters in North Manchuria a highly susceptible population and may account for the severe nature of the epidemics when they arise.

The age incidence has also been studied as well as the susceptibility among persons from different provinces. The results so far obtained provide interesting food for reflection.

TABLE VII. Results of Dick Test in Chinese from Different Provinces.

Result:	Chihli	Fengtien	Kirin	Heilung kiang	Shan- tung	Fukien	Kiang su	Honan	Hupei	Kwang Tung
Dick neg.	287	164	86	12	89	8	7	10	7	9
Dick pos.	199	96	53	5	64	7	6	3	3	5
One plus										
Two „	70	32	12	4	23	1	—	3	2	7
Three „	8	8	5	—	4	—	1	—	—	1
Total	564	300	156	21	180	16	14	16	12	22
% Dick pos.	49.1	45.3	44.8	42.8	50.5	20.0	50.0	37.5	41.6	59.0

TABLE VIII. Dick Test Acc. to Sexes with History of Sc. Fever.

	Negative	Positive	Total	% positive
F.	8	1	9	11.1%
M.	47	3	50	6.0%

TABLE IX. Dick Test Acc. to Chinese Schools, Harbin.

Name of school	Negative	Positive				Total	% positive
		+	++	+++	++++		
Fuchiation							
Mission. Girls' High School							
(age fr. 9-20)	45	20	12	3		80	43.7
Mission. Prim. Girls' School							
(age 7-14)	17	12	14	8		51	66.6
Government Orphanage							
(age 9-18)	92	23	10	6		131	29.7
Commercial School							
(age 15-30)	88	56	10	—		154	45.8
Chi Yeh School							
(age 10-16)	69	32	2	—		103	33.0
Mission. Kinder-garten							
(age 3-6)	3	6	8	6		23	86.1

TABLE X. Dick Test Among Chinese School Boys & Girls:

Age	Negative	Positive	Total	% positive
0-1	—	—	—	—
1-2	—	—	—	—
2-3	—	4	4	100.00
4-5	2	11	13	84.6
6-7	22	52	74	70.2
8-9	38	60	98	61.2
10-11	42	50	92	54.3
12-14	101	48	149	32.8

Another practical application of Dick's toxin is the aid it renders in diagnosing doubtful cases of scarlet fever. A strong positive reaction during convalescence from an illness with a scarlatiniform rash would probably point against the diagnosis of scarlet fever. This test is a valuable adjunct of Schultz and Charlton's blanching phenomenon for later stages of the disease.

Owing to lack of suitable material we cannot at present report upon this test.

In 1911-12 the application of high doses of convalescent serum was introduced by Reiss and Jungmann¹⁶⁾ into the

therapy of scarlet fever. They injected 50-100 cc. intravenously and obtained good results. The convalescent serum had been used since the end of the last century but had always been injected in small quantities. (Leyden, Weissbecker, Huber and Blumenthal, Schultz, a. o.) For that reason only a few good results had been reported.

Many authors (Koch, Bode, Mironescu and Sager, Bernbaum Langer, a. o.) have confirmed the observations of Reiss and Jungmann.

Degkwitz says it is possible to produce temporary immunity against scarlet fever by using 5-10cc. of convalescent serum.⁷⁾

Though the treatment by convalescent serum may be effective, in many cases its wide application is doubtful for it develops full power only at the beginning of the illness and is inefficient against the complications of scarlet fever. Moreover, it is often difficult, especially for small hospitals, to provide convalescent sera from undoubtedly healthy persons.

The investigations of Dochez⁹⁾ are, therefore, heartily welcomed. This author and his associates have introduced a new method of producing an anti-streptococcic serum by immunizing horses with living streptococci after liquid agar had been introduced into the animal. Blake,²⁾ Blake, Trask and Lynch³⁾ Birkhaug¹⁾ have obtained very good results with this serum. We have likewise used this serum provided by Eli Lilly (Minn.) upon patients at Harbin Municipal Hospital, and have seen encouraging results. But the cases are as yet too few to be commented upon.

The investigations of the last few years have produced a new conception of scarlet fever. It may now be considered as a local infection of the naso-pharynx in which a soluble toxin is produced. This is absorbed into the system of the patient where it gives rise to the rash and other constitutional symptoms (quoted by Zingher²²⁾).

This conception supports the advocacy of early local disinfection of the throat. Investigations on *St. haem* lately published by Kuestner state that the virulene of these germs is increased by growing on decaying tissues.¹³⁾ This fact may explain why streptococci have such a destroying power on necrotic tonsils. Probably more stress will be laid in future on the finding of an active agent for throat disinfection (Milne, Elgart¹⁰⁾).

In conclusion, we wish to acknowledge our indebtedness to our Chief, Dr. Wu Lien Teh, for his constant advice and supervision in the course of our researches.

SUMMARY.

1. We have found *Streptococcus haemolyticus* in every one of the 18 cases of scarlet fever investigated. Out of these 18, 15 showed the organism in practically pure culture.

2. The incidence of scarlatina in North China is high, both morbidity and mortality showing a higher percentage than countries of the same latitude in Europe and America.

3. Out of 1275 Dick Tests made on the Chinese population in Harbin, ranging in ages from 1 to over 30 years, we obtained positives in 47.7%. This suggests a higher degree of susceptibility among the Chinese residing in this region.

4. We have also conducted Dick Tests among 542 school children of six schools, and found more positives in the kindergartens (86%) than elsewhere.

5. Investigations made of persons from different provinces of China are as yet too few to be of value, but should be prosecuted on the spot with a view to ascertaining their comparative susceptibility, especially between northerners and southerners.

6. Encouraging results have been obtained from Dochez's serum made in America in the treatment of scarlet fever patients, but we are preparing our own serum, so that it may be tried on a more extensive scale throughout North China.

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DISCUSSIONS.

Dr. T. Murayama: The mortality of scarlet fever here in Tokyo is about 2% in the Municipal Hospital for Infectious Diseases of Tokyo. Generally speaking, the mortality of scarlet fever is getting lower and lower year by year. The most important factor of this fact lies in milder cases, because here in Japan all cases of scarlatina are recorded.

Dr. D. B. Avison: In Korea we have no idea as to the prevalence of scarlet fever because the great majority of cases are not reported to the Health Department. Many doctors would seem to prefer not to subject the patients or the patients' relatives to the discomforts and troubles attendant on such reports and only report such cases as are likely to die when the failure to report would get them (the doctors) into trouble with the authorities.

THE PROBLEM OF VENEREAL DISEASES IN CHINA

BY WU LIEN-TEH

TABLE OF CONTENTS

- A. Introduction.
- B. Information on the Prevalence of V. D. in China.
 - (a) General statements of medical practitioners as to frequency.
 - (b) Incidence of V. D. among patients admitted or treated in hospitals, etc. Frequency of syphilitic changes at *post-mortem*.
 - (c) Special enquiries as to frequency of V. D. infection (past and present) among patients.
 - (d) Incidence of V. D. according to occupation.
- C. Prostitution in certain cities.
- D. Summary of measures taken in Great Britain, France, and U.S.
- E. Discussion of measures possible at present in China.
- F. Recommendations.

A. Introduction

Like most countries of the world China is confronted with the serious problem of venereal diseases. It may be said without fear of contradiction that syphilis at least can only be traced in this country to comparatively recent times. Such an eminent authority as K. Dohi (Dermatological Institute of the Tokyo Imperial University) says that syphilis was imported from the west some time in the 16th century. Our Chinese records prove that it was unheard of in China until the middle of the 16th century, when it was introduced into Canton by Portuguese from India. Syphilis was frequently mentioned in the latter part of the Ming Dynasty (1367-1644), often in an accurate and illuminative manner, and in one interesting monograph written in 1631 the various manifestations and hereditary transmission of the disease are mentioned in full detail.

It was unfortunate from the preventive standpoint that for nearly 100 years after the introduction of western medicine into China venereal disease was, as it were, avoided by responsible

teachers and it was only within recent times, say the last 15 years, that serious attention has been centered upon the two widespread sexual diseases, syphilis and gonorrhoea.

B. Information on the Prevalence of Venereal Diseases in China

I have for a number of years been interested in the public health aspects of this problem in China and studied particularly its connection with prostitution which in this country may be traced to a period even earlier than the hectic days of the Roman Empire. In order to classify the information available regarding the prevalence of venereal diseases in China I have arranged it in five groups:

- a. General statements of medical practitioners as to their frequency.
 - b. Incidence of venereal diseases among patients admitted or treated in hospitals, etc. Frequency of syphilitic changes found at *post-mortem*.
 - c. Special inquiries as to the frequency of venereal infection (past and present) among patients.
 - d. Routine Wassermann Reactions among patients and groups of population.
 - e. Incidence of venereal diseases according to occupation.
- a. *General statements of medical practitioners as to the frequency of venereal diseases.*

Obviously such statements can give only a rough idea of the frequency of venereal diseases, but attention has to be paid to the critical judgement of various authorities. Table I comprises data collected from cities in 8 provinces as well as Hongkong and Formosa which are largely inhabited by Chinese.

The figures show that venereal diseases are rampant everywhere, often to an alarming extent, as much as 50-60 percent of the adult population being affected.

- b. *Incidence of venereal diseases among patients admitted or treated in hospitals, etc. Frequency of syphilitic changes found at post-mortem.*

For these, we have figures from Hongkong (1912-24), Shanghai (1912-25 and 1870-1925) and Manchuria (1913-1925).

In Hongkong the incidence of Syphilis varies from 1.5-3.2% of all admissions into hospitals; in the mortuary the figure is 5%, whilst gonorrhoea claims 0.7-2.5%.

The Shantung Road Hospital, Shanghai, records an average of 6.6% of all admissions between 1870-1925. This Hospital also

records 3.4% male as compared to 2.2% female sufferers from syphilis among out-patients.

Our Plague Prevention Service Hospitals numbering five in North Manchuria record 2.9 to 8.1% (average 6.4%) of Syphilis, whilst Newchwang claims 13.5% of syphilis and 14.8% of gonorrhoea.

The figures collected above probably do not present a high enough incidence of venereal diseases, but a minimum of same for the following reasons:

- i. As far as Inpatients are concerned, the hospitals adopt different policies regarding admittance of venereal diseases; often they refuse admission altogether.
 - ii. Only a portion of patients seek the aid of western hospitals.
 - iii. Only manifest cases are recorded, many of the venereal disease cases being hidden under other headings, like rheumatic, nervous, heart, women's diseases, etc.
 - iv. Traces of former infection may only be accurately detected by such tests as Wassermann.
 - v. Where no such traces are left, careful investigation would reveal history of former infection.
- c. *Special enquiries as to the frequency of venereal diseases (past and present) among patients of hospitals.*

Only one such enquiry is on record (Lennox, China Medical Jl., 1919, p. 326). He says:

Among 4000 married men (poor and middle class)

22.2%	admitted	having	had	gonorrhoea,
6.9%	„	„	„	syphilis,
3.9%	„	„	„	both.

d. *Routine Wassermann Reactions.*

Routine Wassermann Reactions as collected at Soochow, Peking and Shanghai are tabulated in Table Va. The figures vary widely owing to different interpretations by various workers, but it may be said that—as far as our limited figures go—the average percentage of 17.3 follows closely the published records of Great Britain (18.2) and U.S.A. (20.1). See table Va.

e. *Incidence of venereal disease according to occupation.*

In this connection records from Soochow, Peking (3) and Shanghai have been consulted. They show much variation among the different occupations in different localities, but generally it may be said the incidence is highest among soldiers and police,

and lowest among professional people. Business men come rather high, whilst farmers and labourers seem to occupy an intermediate position. In order of scale, the figures show the following: Soldiers and police (over 35%), Business (31.8), unclassified occupation (21.1) farmers (16.8) labourers (15.3), students (13) and professional (7.1). Average 19.5.

C. Prostitution in Certain Cities

In addition to the above a table (Table VIII) deals with the numbers of prostitutes in certain localities. Here it may be interesting to mention that of all cities in China the two (Antung and Taheiho) where there is regular weekly medical inspection of brothel inmates are both situated in Manchuria.

In Canton the proportion of prostitutes to population is 1:837; in Shanghai it is 1:147; in Harbin 1:82; in Taheiho 1:138; and in Newchwang 1:113. The abnormally high percentage in Harbin, an important city with a large floating population, is significant.

D. Short Summary of Measures Taken in Some Countries

It would require too much time if I were to describe in detail the various methods that have been introduced or are being introduced in different parts of the world for the prevention and control of venereal diseases. Suffice it to say that each country with its peculiar national traits proposes to deal with the problem in its own way. For instance reformers in Great Britain with its high moral and religious sense are divided into two main camps, advocating respectively:

- (a) Early treatment (that is, disinfection immediately after intercourse at so-called treatment centers) and
- (b) Prophylaxis (where disinfectants such as 1:3 calomel ointment are employed previous to intercourse). But in view of the frequent unwillingness or inability on the part of individuals to resort to precautions, public opinion in that country is gradually coming round to the view that some sort of compulsion in prophylaxis and notification is necessary. As late as June 16th, 1926, Mr. Basil Peto, M.P., presented a Venereal Diseases Bill 1926 before Parliament to permit the sale by chemists of disinfectants for protection against venereal diseases. In his speech he quoted several medical authorities to prove that early prophylaxis means a lower incidence of venereal diseases. Thus, in one military camp out of 4400 men exposed to infection only 13 cases of infection resulted. In the Naval Gunnery School at Portsmouth between 1918-20,

923 bottles of 1:1,000 Permanganate solution were supplied to men exposed to infection, and only one case of disease resulted. At the Royal Artillery Barracks at Portsmouth in 1917, 3750 soldiers received early disinfection with only five casualties. There is no reason why similar beneficial results may not be expected amongst the civil population. Statistics show that a large proportion of paralysis, malformation, mental deficiency, insanity, epilepsy and blindness affecting greatly the efficiency of the population is traceable to syphilis and gonorrhoea. For the treatment of patients suffering directly or indirectly from these two diseases the Government spent at least £600,000 in the year 1924-25. In Great Britain the Venereal Diseases Act of 1917 is still in force, by which treatment of venereal diseases is prohibited to all except duly qualified medical practitioners. In the New Venereal Diseases Bill of 1926 it is proposed to extend certain rights to chemists as well as doctors in the matter of prophylaxis. Dr. A. W. Pope has suggested the following measures against venereal diseases for Great Britain (Lancet, June 26th, 1926):

a. Duty of Person Infected.—(1) Every person suffering from any form of venereal disease as soon as he is aware or has reason to believe that he is suffering from such disease, shall forthwith consult a medical adviser with respect thereto, and shall furnish to him his correct name and address and place himself under his treatment. (2) Every such person shall continue to attend or be attended by his medical adviser, and to follow his advice and treatment until he is deemed free from infection. (Provision to be made for change of medical adviser and for routine procedure in case of neglect to continue treatment.) (3) No person shall knowingly or wilfully infect any other person with venereal disease or do not permit any act likely to lead to such infection.

b. Duty of Parent or Guardian.—Every parent, guardian or person in charge of child (under 16 years of age) or mental defective suffering from any form of venereal disease, and who knows that such child or defective is suffering from such disease, shall cause the child or defective to be treated and continue treatment for such disease by a medical adviser.

c. Penalties.—Section 1 (3) of the Public Health Act, 1896, provides that if any person willfully neglects or refuses to obey the execution of any regulation under Section 130 of the Public Health Act, 1875, he shall be liable to a penalty not exceeding £100 and in the case of continuing the offence of a further penalty of £50 per day.

d. Duties of Medical Practitioner.—(1) The doctor shall direct the patient's attention to the infectious character of the disease, and to the necessity of continuing treatment until free from liability to infect, and to the penalties prescribed. (2) To arrange for transfer to another medical adviser when the patient so desires. (3) When a patient discontinues medical treatment without adequate reason, the medical adviser will forward his name and address to the medical officer of health. In any case where a fee is not paid by the patient to the doctor, the provision for such payment shall be made on the lines which the Ministry have already authorised in Liverpool in regard to the domiciliary treatment of non-insured tuberculous persons. Suitable forms shall be provided for the use of medical practitioners intimating the obligations upon patients—arrangements for transfer when necessary—and forms of notice to the medical officer of a health as to acceptance of a patient by the practitioner, and when necessary, his non-attendance. Drugs specially necessary for treatment shall be provided free of charge to medical men as hitherto. Ordinary prescriptions shall be paid for as in the scheme for domiciliary treatment of tuberculosis.

c. Duty of Medical Officer of Health.—The medical officer of health, on receipt of a notice from the medical adviser in regard to any patient, shall make inquiries from the person named as to the reason for discontinuance of treatment, and unless satisfied shall cause an information to be laid in a court of summary jurisdiction.

Turning to *France* we find that the problem of venereal diseases has become so serious that very radical laws are proposed, dealing especially with syphilis. These include compulsory treatment by physicians, notification by physicians of the patient's condition to the proper authorities as to the source of infection as well as the stage of the disease, with heavy fines in case of non-observance.

In *America* each of the 48 States of the Union has its own laws against venereal diseases. Even in the matter of prostitution they vary considerably, some cities regulating the vice whilst the majority prohibiting it altogether. Thanks to the force of public opinion it is possible that before very long the few red-light districts still existing will be abolished, but this does not mean that venereal diseases will be entirely stamped out. In Chicago large numbers of women are every morning hauled up before the Court for practising illicit prostitution. Two women physicians are employed to examine the delinquents and if found diseased they are sent to a special ward in the Municipal Hospital. In the city of Detroit one of the most important clinics is that presided by Dr. R. S. Dixon, a keen man with initiative who, although the law nominally prohibits prostitution, manages to

have hundreds of women visiting him daily at his office to voluntarily register themselves and receive treatment when diseased. Dr. Dixon actually quizzes them over their methods of preventing infection in the course of their occupation and gives demonstrations on the proper technique to be adopted. In order to aid local authorities and social organisations in their anti-venereal campaign the U.S. Public Health Service has published an interesting booklet of 60 pages for the guidance of community leaders. There are three major chapters:

I. Educational Measures on:

1. Arousing the community to the seriousness of venereal diseases.
2. Centering attention on the communicable nature of gonorrhoea and syphilis.
3. Emphasizing importance of prevention.
4. Education against the danger and degradation of prostitution.
5. Such education to reach all classes of the population.

For broadcasting information, educational methods are recommended in the form of pamphlets, books, lectures, exhibits, lantern slides, motion pictures, radio talks, etc.

II. Medical Measures, emphasizing particularly:

1. Treatment should be free or for a nominal fee.
2. Treatment should be easy of reach.
3. Treatment should be prompt and continuous.
4. Treatment should be scientific.

For carrying these out there are recommended V.D. Clinics, hospital treatment, venereal diseases control ordinances, exposing quacks and quack medicines, and extensive use of laboratory facilities.

III. Legal Measures by means of:

1. Laws prohibiting prostitution,
2. Indirect attack on prostitution, such as injunction of property.
3. Elimination of clandestine prostitution, such as control of rooming houses and hotels, licensing public dance halls, licensing taxicabs.
4. Disposition of prostitutes, e.g. treating them when sick, giving them work, etc.
5. Compulsory control of ophthalmia neonatorum.
6. Marriage reform, e.g. requiring medical certificates from contracting parties.

E. Discussion of Measures Possible at Present in China

How far are these more or less ideal laws applicable to China at the present moment? In the course of my experience I have come across women-teachers who demand certificates from me guaranteeing the good health of their proposed husbands before they are willing to enter into matrimony. On the other hand I have seen well educated families who persist in uniting their beloved daughters to obviously diseased young men in spite of medical advice. The poor as a rule have little or no choice though it must be said that the majority of country peasants of whom the bulk of China's population consists, are free from venereal diseases.

It will be a long time before prostitution can be abolished in China as a result of public agitation and sentiment.

Almost every Chinese city now has a few western trained practitioners whose main income is derived from the injection of Salvarsan (or substitute) solutions into their patients. Harbin alone with its 300,000 inhabitants has over 200 such "hospitals." It pays such practitioners better to charge \$20-30 for each injection than to advise on prophylaxis bringing small monetary returns. In the latter case the packet costs only 30-50 cents and does not immediately advertise his skill in treatment.

Nation-wide health education is absolutely necessary if the present alarming incidence of venereal diseases is to be diminished. I would advise that the medical profession of China join in this crusade against ignorance, and advocate without hesitation in homes, college institutions and hospitals, not only the need of control of the sexual appetite but also efficient prophylaxis. May I summarise these proposals?

F. Recommendations

- (1) Emphasize prevention of sexual diseases as well as other aspects of health education, such as infant welfare, infectious diseases, tuberculosis, etc.
- (2) Prepare and distribute popular health tracts on venereal diseases, and utilise all reliable agencies for the dissemination of knowledge.
- (3) Invite co-operation of mission and lay teachers in this publicity work.
- (4) Prepare and sell at cheap prices prophylactics of proved value, such as calomel cream, permanganate, etc.
- (5) Provide as cheap treatment as possible for all forms of venereal disease in hospitals, so that poor and rich may be equally benefitted.

For this purpose, the location and time of the clinics should be adapted to individual needs.

TABLE I.

Locality.	Reference.	Year.	Incidence of V.D. Remarks.
Antung (Manchuria)	Larsen, Ch. M. Jl. 1919, p. 280.	1917-18	"It seems as if at least half the population is infected."
Canton (Kwangtung)	Reynolds, <i>ibid.</i> , 1917, p. 225. Li Ting-An. Nat. Med. Jl., 1925, p. 324.	1915-16 1924	Both syphilis and gonorrhoea extremely prevalent. Syphilis is one of the most important causes of morbidity and mortality.
Hainan (Kwangtung)	Bercovitz, <i>ibid.</i> , 1924, p. 788.	1924	Syphilis one of the commonest diseases. Estimates 50-60% of population affected.
Pakhoi (Kwangtung)	Baronsfeather, <i>ibid.</i> , 1917, p. 443.	1916	Syphilis and gonorrhoea among the most common diseases.
Yeungkong (Kwangtung)	Debson <i>ibid.</i> , 1911, p. 71.		Syphilis: "I am compelled to believe, that seven-tenths of all patients present a lowered vitality through this venereal disease."
Changsha (Hunan)	Jen, Ch. Med. Jl., 1912, p. 359. Hume, <i>ibid.</i> , 1917, p. 309.	1911 1914-15	"Tuberculosis and syphilis still heading the list of diseases prevalent among the Chinese..... V.D. very common.
Hunan	Pakes, Ch. Med. Jl., 1911, p. 362.		"The extraordinary prevalence of s in all its stages and manifestations is enough to stagger one." Go. norrhoea not so often seen.
Chinkiang (Kiangsu)	Bradshaw, <i>ibid.</i> , 1919, p. 184 and 1920, p. 86.	1917-18 1918-19	V.D. fairly common.
Soochow (Kiangsu)	Russell and Park, <i>ibid.</i> , 1918, p. 69.	1916	See special table. V.D. appear to be on the increase.
Shanghai (Kiangsu)	Hou, Nat. Med. Jl., 1925, p. 27.	1923	See special table. Speaks of steady increase of V.D.
Chungking (Szechuen)	Post, Ch. Med. Jl. 1917, p. 260.	1913-14	In the German Poliklinik there were 521 cases of syphilis (including 30 congenital) and 176 cases of gonorrhoea among 4787 total cases (10.9 & 3.5% resp.)
Formosa	Maxwell, <i>ibid.</i> , 1915, p. 28.	1913	"Venereal disease is rampant Syphilis is a commoner and much more severe disease than is now ordinarily met with in England. Amount of acquired syphilis in children is simply appalling..."

Locality	Reference	Year	Incidence of V. D. Remarks
Hongkong	Medical Reports.		See special table. Increase in numbers of patients 1921-23. In 1924 no further increase.
Ichang (Hupeh)	Borthwick, Ch. Med. Jl. 1917, p. 31.	1914-15	"Day in and day out, we are brought face to face with V. D."
	Graham, <i>ibid.</i> , 1918, p. 70	1916-17	"Patients with V. D. are seen in great numbers..."
	<i>Ibid.</i> , 1919, p. 183.	1917-18	"Venereal diseases have been even more in evidence than formerly owing to the large number of troops in the district....."
Kansu	King, <i>ibid.</i> , 1925, p. 20.		Syphilis "widespread. Some of the worst cases come from among the Thibetans..."
Tengyueh (Yunnan)	Ram Lall Sircar, Ch. Med. Jl., 1912, p. 247.	1912	V. D. among the most prevalent ones
	Chose, <i>ibid.</i> , 1920, p. 87.	1918-19	
Wenchow (Chekiang)	Stedford, <i>ibid.</i> , 1916, p. 338.	1914	Syphilis "extremely common."

TABLE II. SYPHILLS AND GONORRHOEA ADMISSION, HONGKONG HOSPITALS.

Date	Civil Hosp.				Goal Hosp.				Kowloon Disp.				Tung Wa Hosp.				Victoria Mort.				Kowloon Mort.				Total			
	Syph No.	%	Gon No.	%	Syph No.	%	Gon No.	%	Syph No.	%	Gon No.	%	Syph No.	%	Gon No.	%	Syph No.	%	Gon No.	%	Syph No.	%	Gon No.	%	Syph No.	%	Gon No.	%
1912	75	2.8	50	1.9	5	0.4	1	0.1	74	2.6	138	4.9	195	4.7	29	0.7	21	0.8	7	0.5	377	2.6	218	2.0
1913	70	2.6	51	1.9	8	0.3	2	0.1	70	2.8	80	3.2	152	3.2	78	1.7	21	0.8	0.	443	2.9	211	2.0
1914	74	2.8	56	2.1	10	2.7	2	0.4	97	3.3	102	3.5	141	3.1	49	1.7	101	4.5	1	0.1	424	3.0	209	2.0
1915	75	2.5	49	1.6	4	1.7	2	0.5	290	5.4	230	4.3	87	1.9	36	0.8	234	10.6	6	0.6	696	4.2	317	2.4
1916	98	3.3	46	1.5	2	0.8	1	0.4	222	3.1	284	4.0	128	2.4	21	0.4	296	8.0	5	0.4	751	3.6	352	2.3
1917	130	4.0	54	1.7	3	1.8	2	1.1	190	2.1	353	3.8	117	2.3	36	0.7	251	8.0	2	0.1	693	3.1	445	2.5
1918	86	2.4	65	1.8	6	2.0	0	0	252	1.9	202	1.5	105	1.7	48	0.8	357	8.7	4	0.2	810	2.7	315	1.3
1919	125	3.2	125	3.2	8	?	0	0	216	1.6	260	2.0	74	1.1	18	0.3	119	3.8	5	0.3	547	1.9	403	1.7
1920	144	3.1	176	3.8	10	3.4	2	0.8	205	1.9	249	2.3	158	2.2	29	0.4	317	8.2	9	0.6	843	3.0	456	2.0
1921	217	4.6	149	3.1	7	3.0	2	0.8	221	1.4	160	1.0	249	3.6	42	0.6	152	4.4	14	1.0	860	2.7	353	1.1
1922	172	4.0	135	3.1	7	1.0	9	2.5	264	1.1	215	0.9	349	4.3	61	0.7	54	1.4	29	1.4	875	2.1	420	1.2
1923	177	3.3	113	2.1	6	2.0	2	0.7	353	2.4	96	0.7	575	5.7	72	0.7	65	1.3	70	3.1	1246	3.3	283	0.9
1924	169	2.8	125	2.1	1	0.2	5	1.2	517	2.6	138	0.7	427	4.5	57	0.6	99	2.5	113	4.3	1326	3.1	325	0.9
Average %	...	3.2	...	2.3	...	1.5	...	0.8	...	2.5	...	2.5	...	3.1	...	0.6	...	5.0	1.0	3.0	...	1.7

TABLE IIIA. SYPHILIS AND GONORRHOEA AND CHANCROID ADMISSIONS, SHANGHAI HOSP.

Date	Isolation Hosp. (Chinese cases only)						Goal Hosp. (All cases)						Police Hospital (Chinese cases only)						Total					
	Syph. No.	%	Gon. No.	%	Ch. No.	%	Syph. No.	%	Gon. No.	%	Ch. No.	%	Syph. No.	%	Gon. No.	%	Ch. No.	%	Syph. No.	%	Gon. No.	%	Ch. No.	%
1912	14	1.3	292	28.3	15	1.4	0	0	94	12.0	77	9.7	16	2.1	80	10.7	80	10.8	30	1.2	466	18.1	173	6.7
1913	0	0	267	28.5	8	0.9	15	2.9	49	9.3	36	6.9	19	2.4	57	7.2	69	8.9	34	1.5	373	16.7	119	5.3
1914	0	0	204	20.8	1	0.1	9	0.7	107	7.9	94	6.9	33	3.5	84	8.8	82	8.6	42	1.2	395	12.0	177	5.4
1915	2	0.2	236	25.3	6	0.6	1	0.1	104	6.1	46	2.7	12	1.1	76	7.4	75	7.3	15	0.4	416	11.4	127	3.5
1916	1	0.1	306	44.2	13	1.9	4	0.2	77	4.6	114	6.8	9	1.0	37	4.0	29	3.1	14	0.4	420	12.7	156	4.7
1917	5	0.6	287	35.1	3	0.4	4	0.2	103	4.0	166	6.6	9	1.1	32	4.0	20	2.5	18	0.4	422	10.0	189	4.5
1918	2	0.4	146	26.2	4	0.7	20	1.0	78	3.8	180	8.8	10	1.1	35	3.9	18	2.0	32	0.9	259	7.4	202	5.7
1919	10	1.7	110	19.1	3	0.5	29	1.5	91	4.8	191	10.1	8	0.9	24	2.8	11	1.3	47	1.4	225	6.8	205	6.2
1920	2	0.4	53	11.4	1	0.2	27	1.0	132	5.2	140	5.5	11	1.3	26	3.0	12	1.4	40	1.0	211	5.4	153	3.9
1921	—	—	—	—	—	—	146	9.3	126	8.0	76	4.8	52	6.4	27	3.3	8	1.0	198	8.3	153	6.4	84	3.5
1922	—	—	—	—	—	—	71	2.8	240	9.3	130	5.0	50	5.3	35	3.7	14	1.5	121	3.4	275	7.8	144	4.1
1923	—	—	—	—	—	—	121	4.8	348	13.8	147	5.8	184	18.7	34	3.4	10	1.0	305	8.7	382	10.9	157	4.5
1924	—	—	—	—	—	—	115	3.8	378	12.4	165	5.4	46	5.3	37	4.2	10	1.1	161	4.1	415	10.5	175	4.4
1925	—	—	—	—	—	—	87	2.7	393	12.4	140	4.4	17	1.7	41	4.1	10	1.0	104	2.5	434	10.4	150	3.6
Average %		0.7		26.5		0.7		2.4		8.1		6.4		3.7		5.0		3.7		2.5		10.5		4.7

TABLE IIIB. V. D. CASES IN SHANGHAI HOSPITALS.

Years	General Hos- pital (almost exclusively foreigners)		Shantung Road Hospital (Chinese)			St. Luke's	
	No.	%	No.	%	Remarks :	No.	%
1865-69	450	19.2					
1870-74	289	16.5	3329	5.3	Total new cases.		
1875-79	232	14.1	3869	5.2	Total new cases.		
1880-84	214	10.0	4511	5.9	Total new cases. No figures available for 1883.		
1885-89	203	8.7	5310	5.7	Total new cases. No figures available for 1885.		
1890-94	242	8.3	5627	5.9	Total new cases. No figures available for 1893.		
1895-99	402	9.3	24004	6.9	Total cases (old and new).		
1900-04	616	9.3	35251	7.6	Total cases (old and new).		
1905-09	602	7.9	345	5.4	Only in-patients.		
1910-14	599	7.5	458	5.7	Only in-patients.		
1915-19	507	6.6	385	4.2	Only in-patients.	1919 195	8.4
1920-22	591	8.2	262	4.9	Only in-patients.		
1923	—	—	2555	6.3	Only out-patients.	652	?
1924	—	—	3023	6.2	Total new cases.		
1925	—	—	2642	6.8	Total new cases.		
Total	4947	9.1	91571	6.6			

TABLE IIIC. SHANTUNG ROAD HOSPITAL OUT-PATIENTS SHANGHAI.
SYPHILIS.

Year.	Male		Female		Total	
	No.	%	No.	%	No.	%
1923	952	3.1	287	3.0	1239	3.0
1924	1034	3.1	261	2.1	1295	2.8
1925	1033	4.1	163	1.5	1196	3.3
Total	3019	3.4	711	2.2	3730	3.0

GONORRHOEA.

Year.	Male		Female		Total	
	No.	%	No.	%	No.	%
1923	826	2.6	38	0.4	864	2.1
1924	997	3.0	91	0.7	1088	2.4
1925	913	3.6	54	0.5	967	2.7
Total	2736	3.0	183	0.5	2919	2.4

CHANCROID.

Year.	Male		Female		Total	
	No.	%	No.	%	No.	%
1923	428	1.4	24	0.2	542	1.1
1924	470	1.4	25	0.2	495	1.1
1925	355	1.5	17	0.2	372	1.0
Total	1253	1.4	66	0.2	1319	1.1

TABLE IV. SYPHILIS AND GONORRHOEA, PLAGUE PREVENTION HOSPITALS, MANCHURIA.

Date	Harbin				Tahaiho				Sansing				Lahasusu				Manchouli				Total				% of V. D. of Newchwang Hospital	
	Sypb. No.	%	Gon. No.	%	Sypb. No.	%	Gon. No.	%	Sypb. No.	%	Gon. No.	%	Sypb. No.	%	Gon. No.	%	Sypb. No.	%	Sypb. No.	%	Gon. No.	%	Sypb.	Gon.		
1913	639	5.6	114	1.0	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
1914	1273	7.1	203	1.1	131	11.6	36	3.2	65	1.4	81	1.7	—	—	—	—	—	—	—	—	—	—	—	—	—	
1915	1399	8.9	273	1.7	137	6.9	31	1.6	191	9.1	99	5.2	42	3.7	18	1.6	—	—	—	—	—	—	—	—	—	
1916	1334	5.11	149	1.3	108	4.4	62	2.5	53	1.7	45	1.4	4	3.8	17	1.6	—	—	—	—	—	—	—	—	—	
1917	746	7.0	241	2.2	309	8.1	309	8.1	29	0.7	48	1.2	40	4.2	7	0.7	—	—	—	—	—	—	—	—	—	
1918	387	4.0	104	1.1	505	7.7	339	5.2	70	1.8	55	1.4	45	7.2	12	1.9	—	—	—	—	—	—	—	—	—	
1919	332	3.7	135	1.5	583	8.1	372	5.2	117	3.8	27	0.9	24	6.9	9	2.6	—	—	—	—	—	—	—	—	—	
1920	755	8.7	297	3.4	821	11.6	307	4.3	185	4.5	45	1.1	79	4.3	30	1.6	—	—	—	—	—	—	—	—	10.0	
1921	343	7.9	52	1.2	452	9.3	290	6.0	154	1.3	50	1.4	187	9.1	109	5.3	—	—	—	—	—	—	—	—	12	
1922	583	6.6	134	1.5	128	4.4	43	1.5	91	2.1	51	1.2	90	6.1	13	0.9	175	8.0	7.9	7.9	1067	5.4	412	2.1	15	
1923	772	4.6	428	2.6	150	4.0	65	1.7	78	1.4	47	0.9	19	1.4	0	0	484	11.0	4.3	4.3	1503	5.1	646	2.1	20	
1924	976	6.4	404	2.6	109	3.7	45	1.5	65	1.5	13	0.3	57	3.1	18	1.0	182	8.0	5.5	5.5	1389	5.2	606	2.3	18	
1925	1564	7.3	531	2.5	77	3.0	39	1.5	114	2.8	37	0.9	81	4.9	58	3.5	174	5.7	5.1	5.1	2010	6.1	820	2.5	14	
Average %		6.9		1.8		6.9		3.5		2.9		1.5		5.0		1.9		8.1		5.7		6.4		2.3		10

TABLE VA. INCIDENCE OF WASSERMANN POSITIVES AMONG CERTAIN GROUPS.

Locality	Reference	Category of exd.	Male		Female		Total		Married		Single		Age									
			No. exd.	%.	No. exd.	%.	No. exd.	%.	No. exd.	%.	No. exd.	%.	No. exd.	%.	No. exd.	%.	No. exd.	%.	No. exd.	%.	No. exd.	%.
Soochow	a) Snell and Chang, Ch. Med. J., 21/36.	In-pt.											1-9	10-19	20-29	30-39	40-49	50-59	No. exd.	%.	No. exd.	%.
			586	43.2	166	24.1	752	39.0	660	40.0	92	30.4	11	18.1	75	29.3	252	37.7	199	42.7	140	47.8
Peking	Korns. Ch. Med. J., 20/624.	Domest. Serv.																				
			301	12.6	60	3.3	361	11.0	278	11.1	83	10.8										
Peking	b) Sia, Med. J., 21/39.	Med. In-pt.											1-10	11-20	21-30	31-40	41-50	51-60	Over			
			494	24.1	8	12.5	502	25.9	327	26.3	175	19.4	11	0.0	58	5.2	174	16.1	137	38.7	71	36.6
Peking	Tsen, Ch. Med. J., 20/159.	Pat. and Serv.																				
			1318	16.1	318	9.43	1636	14.79	1011	15.4	423	c) 12.8	18	0.0	151	3.3	450	13.4	310	23.5	146	23.3
Peking	Korns. Ch. Med. J., 21/382.	Domest. Serv.																				
			608	12.0	105	5.7	713	11.0														
Peking	Tso, Ch. Med. J., 23/226.	Hosp. Employ.																				
							953	8.0														

Remarks : (a) The one-two-and three-plus reactions were all regarded here as positive, whereas Tsen considers only fourplus reactions as positive.
(b) Foreign patients are included in these statistics. (d) Age unknown in 469 cases with 57 positives (12%)
(c) Sex unknown in 202 cases with 32 positives (16%) (e) Tsang (Nat. Med. J., 1925, p. 173) writes:
"Out of 1797 Wassermann reactions done at the St. Luke's Hospital, Shanghai in 1923 (75% being done as a routine for the inpatients of the Hospital), 438 gave positive reactions, i.e. 30%".

TABLE VB. EVIDENCE OF WASSERMANN POSITIVES IN CERTAIN COUNTRIES.

Country.	No. exd.	% +	Categories exd.		
China	4917	17.3	Patients & healthy persons (see table Va)		
France	1040	38.5	With chronic dis.	608	(48.6% +)
			Insane	329	(26.7% +) ×
			Maternity c.	103	(15.5% +)
× Great Britain	4613	18.2	Gen. Hosp. pt.	1483	(20% +)
			" " "	1435	(10.2% +) ×
			Insane	552	(22.8% +) ×
			Maternity c.	271	(24.7% +)
			Nervous dis.	122	(41.0% +)
			Eye dis.	250	(50.0% +)
			Healthy pers.	500	(9.4% +)
× U.S.A.	18458	20.1	Gen. Hosp. pt.	10935	(26.4% +)
			" " "	484	(12.0% +) ×
			Insane	3000	(15.0% +)
			Maternity c.	1839	(2.5% +)
			Med. & neur. c.	1700	(12.0% +)
			Med. c.	500	(12.8% +)

Remarks:

The figures for France and Great Britain are culled from Vedder, Syphilis and Public Health, 1918; those for U.S.A. from Tse, China Medical Journal, 1923, p. 228.

* Means cases with manifest syphilis or metasymphilis deducted.

TABLE VI.
(Incidence of V. D. according to occupation).

	Soochow	Canton	Peking (a)	Peking (b)	Peking (c)	Shanghai	Total
	Snell and Chang Ch. M. Jl., 1921, p. 36	Oldt. Ch. M. Jl., 1932, p. 776	Tso, Ch. M. Jl., 1923, p. 226	Tsen Nat. M. Jl., 1920 p. 159	Sia, M. Jl., 1921 p. 39	Tsang, Nat. M. Jl., 1925 p. 172	(Exclusive of Tsang's figures)
	Routine Wasser. Test. In-pts.	Clin. diag. V. D. In-pts	Wasser. React. in Hosp. employees	Wasser. React. in pts. and serv.	Wasser. React. In-pts.	Clin. diag. primary syphilis in outpats.	
	No. exd.	No. exd.	No. exd.	No. exd.	No. exd.	No. exd.	No. exd.
	% +	% +	% +	% +	% +	% +	% +
Soldier, Officers	58 40.0	235 44.7	— —	199 25.12	117 23.3	— —	609 35.8
Police	19 47.4	— —	— —	— —	— —	— —	19 47.4
Farmer	208 34.1	244 4.4	— —	66 7.57	31 16.1	— —	549 16.8
Laborers	98 40.8	243 23.0	746 9.2 Mechan. Cooks. coolies helpers	698 14.18	101 20.1	55 52.0	1886 15.3

Business	69	52.1	183	38.0	—	—	174	18.96	63	33.3	26	24.5	516	31.8
Student	46	37.0	117	11.4	<div> { <div>clerks, nurses, dressers technician</div> <div>192</div> <div>3.1</div> </div>		<div> { <div>Learned class</div> <div>212</div> <div>5.66</div> </div>		76	2.6	<div> { <div>Learned class</div> <div>8</div> <div>7.5</div> </div>		239	13.0
Profes- sional	37	35.0	—	—					55	7.0	8	7.5	476	7.1
House wife	68	26.4	—	—					—	—	—	—	68	26.4
Miscell- aneous	38	30.0	—	—					41	31.7	—	—	85	31.8
No special work	85	47.6	Children 122 0						38	28.9	—	—	246	21.1
Not Recorded	—	—	—	—	15		281		—	—	17	16.0	296	13.8
Total	754	39.0	1144	21.3	953	8.0	1636	14.79	502	23.9	106	10.0	4989	19.5

TABLE VII. PROSTITUTION IN CERTAIN CITIES IN CHINA

Locality	Reference.	Year.	No. prost.	Per capita. of Pop.
Canton	Oldt, Ch. Med. Jl., 1923, p. 776.	1921	1936	1:837
Shanghai (Intern. Rep. of Vice Comm., Settl.)	Nat. Med. Jl., 1920, p. 126.	1871	1612	?
		1919-20	4522	1:147
Harbin		1911	300	1:80
		1921	3412	1:41
		1926	1459	1:82
Taheiho		1926	116	1:138
Newchwang		1926	967	1:113

MEDICAL PROGRESS IN CHINA SINCE THE REPUBLIC

BY WU LIEN-TEH

In spite of the disturbed state of the country since the establishment of the Republic in 1911, the progress of medical and sanitary science has been very marked and steady. This has shown itself in two ways: (1) the readiness with which both the Central Government and the provincial authorities have established and supported hospitals and institutions of a sanitary nature; and (2) the increased interest shown by the general public in Western medicine, home cleanliness, and matters bearing a hygienic value.

The era in modern preventive medicine may be said to have commenced in 1911, when the Chinese Government and people noticed the great superiority of accurate scientific methods, as compared with crude haphazard methods for the suppression of the pneumonic plague in Manchuria. On one hand, they saw a body of clean, earnest, and fearless young men equipped with knowledge, microscopes, instruments and disinfectants and succeeding in their work of suppressing the epidemic; on the other, they encountered talkative, old-fashioned, uncertain men, whose one idea was to needle and drug the patients and who, because they took no precautions against the disease, died as easily as the patients. One great result of this epidemic was the establishment of the North Manchurian Plague Prevention Service, consisting of hospitals at Harbin, Tsitsihar, Sansing, Newchwang, Taheiho, Manchouli, and Lahasusu. Harbin, being the commercial center of North Manchuria and also the principal focus of infection in 1911, has the largest hospital, covering an area of six English acres and containing an up-to-date bacteriological laboratory capable of handling all sorts of bacteriological research. In ordinary times, when no epidemic exists, these hospitals are used for general purposes, surgical operations being performed and inpatients suffering from various ailments being admitted for treatment. The Medical Staff of the Plague Prevention Service consists of ten male and two female doctors distributed among the different hospitals. Inside the hospital at Harbin, there is a fine medical library, much patronized by the American and other foreign doctors of this city. An interesting museum of

medical objects is also attached to it. Besides annual medical reports, the Plague Prevention Service has published two voluminous scientific reports containing research and other matters carried out by members of the Service. Now and then, as during the last summer, another epidemic, namely cholera, claimed the attention of the staff. Although very severe, it was suppressed within six weeks with the low mortality of fourteen per cent, as compared with a mortality of fifty to sixty per cent in other cities, where the outbreak did not subside until the end of November.

In November, 1913, a Presidential mandate was issued authorizing the dissection of dead bodies in China. This, together with the imperial sanction of cremation of cadavers from plague in 1911, has undoubtedly paved the way further for medical progress in China. Another interesting landmark in the annals of medicine in this country, was the foundation of the National Medical Association in 1914, when thirty medical graduates from European, American, Japanese, and Chinese colleges met together in Shanghai and formed the nucleus of the Association. It has now a membership of over 450 male and female members. The first President was Dr. Yen Fu-ching, M.D., of Harvard and the second Dr. Wu Lien-teh. So far, two conferences of the Association have been held, once in 1915 (Shanghai) and once in 1916 (Canton). The third one will be held this year (1918) in Peking in conjunction with the Medical Missionary Association, at which it is expected famous anatomists, physiologists, and specialists will attend from various parts of the world.

On September 30, 1915, an important mandate was issued recognizing Western medicine as the proper standard of medical practice in China. Any one practicing medicine in the country, will henceforth be required to produce a certificate from the Police. It would be better if this power were centered in the hands of a recognized Central Medical Council, but until that comes into being, the police permit will have to stay.

Up to 1915, Peking had no proper hospital for the treatment of infectious disease, such as scarlet fever, diphtheria, typhoid, etc. Thanks to the liberal policy of the then Minister of Interior (Mr. Chu Chi-chien), a hospital for this purpose was built on tenth street, North City, capable of accommodating sixty beds. This hospital has done creditable work under the combined direction of Yen Chi-chung (graduate of Tokyo University) and Dr. S. P. Chen (M.B. of Cambridge). Cases of small-pox are now sent to the Auxiliary Epidemic Hospital in the grounds of the Temple of Heaven outside Chien Men.

The year 1914 saw the visit to this country of a commission of medical experts appointed by the Rockefeller Foundation to

consider the possibility of establishing two first-class medical colleges in China for the training of future Chinese doctors. Another commission followed in 1915, and as a result it was decided to found two well-equipped colleges and hospitals, one in Peking and the other in Shanghai. The one in Peking is now nearly completed and will be formally opened in the fall of 1920. The cost of this place has approximated five million gold dollars, and it is expected that the annual budget will not be less than one million, though only one hundred students will be trained at one time. When finished, and in working order, the Rockefeller Medical College and Hospital of Peking will eclipse anything in existence in Europe or Asia in the magnificence of its architecture and the completeness of its outfit.

The Peking Central Hospital, upon which the writer devoted the best part of four years in collecting funds, constructing and equipping, was finally opened on January 27, 1917. The total cost is \$300,000 and it is intended to serve as a model hospital for every Province in China. It is modeled upon the American plan and possesses three stories and a basement. It contains four large wards, each accommodating twenty-five beds as well as a series of fine private rooms for first and second class patients. Until the opening of the Rockefeller Hospital the Central Hospital will stand as the finest institution of its kind in China or Japan.

As a result of the second visitation of pneumonic plague in Shansi, China, in 1918, the Central Government devoted the balance of the one million loan (obtained for the suppression of the epidemic) to the establishment of the Institute for Infectious Diseases in the grounds of the Temple of Heaven—a beautiful spot covered with pine trees. Here a series of isolated red brick buildings have been erected for the different departments of the Institute, e.g., Investigation, Serumtherapy Veterinary, Chemical, etc. Over forty experts, mostly graduates of Japan, are connected in one way or another with the Institute. During the last summer, when cholera threatened Peking and the vicinity, this Institute distributed vaccine made in its laboratories for use among the troops. The Chemical Department undertakes to analyze patent medicines and chemicals submitted to it for a fixed fee. The Institute is connected with the Ministry of Interior and comes directly under the Department of Sanitation.

The first Medical College established in North China is the Peiyang Medical, now Naval Medical College, founded in 1893 by Viceroy Li Hung-chang in remembrance of the services rendered to his family by the British missionary Dr. Mackenzie.

After Dr. Mackenzie's death, several past students of the College were appointed Directors, of whom the most prominent was Dr. W. T. Watt. The present staff consists of Dr. H. Y. King (Director), four French, one British, and ten Chinese professors. The Army Medical College was started in Tientsin in 1903 by Viceroy Yuan Shih-k'ai, the first Director being Dr. Hsu Hua-ching, one of the first four graduates of the Peiyang Medical College. In 1918, this College was transferred to Peking. As at present constituted, it occupies an enormous area in the east city between Fifth and Tenth Streets, the ground comprising nearly forty acres. Besides the College and Hospital, there will be built the Veterinary College and the Military Drug Factory. The whole place is under the efficient management of Surgeon General Chuan Shao-ching. The total cost is in the neighbourhood of \$800,000.

A noteworthy feature of this medical progress is the increasing co-operation of Chinese and foreigners. Instead of adopting an exclusive policy, missionary institutions are inviting more and more the assistance of their Chinese colleagues. This broad-mindedness is a wise one, for the natural teachers, healers, and research workers in China are the Chinese themselves, many of whom have now received first-class training abroad. In this respect, the China Medical Board of the Rockefeller Foundation is more far-seeing than the authorities of the Hongkong University, for it has already on its staff at least twelve Chinese doctors out of a total of thirty-three teachers.

Other institutions which have contributed considerably to the progress of medicine in China are the Yale Medical School in Changsha (Hunan); the Union Medical Department of the Shantung Christian University (Tsinanfu); the Kung Yi Medical College and Hackett Medical College (both in Canton), at the latter women receive a good training; the French Medical College in Canton with which is affiliated the Hospital Doumer; the Sino-French Medical College at Changtehfu (Honan) to which is attached a small Institut Pasteur; and the Medical Department of the French Catholic University of Aurore in the French Settlement of Shanghai.

One institution, unique in its way, has perhaps done more than any individual school for the propagation of hygienic ideas among the masses. This is the famous Tsinanfu Institute, founded in 1905 by the Reverend J. S. Whitewright of the English Baptist Mission. The buildings cover over three English acres and have cost over \$150,000. About half a million persons visit the Institute in the course of one year. There should be at least one such institute in every large city in China for the training of the masses in modern progress, including hygiene.

An interesting advance has been made in the appointment of a Terminology Committee, consisting of representatives of the Ministry of Education, National Medical Association, Medical and Pharmaceutical Society, Medical Missionary Association, and Kiangsu Educational Association, to determine the terms to be used throughout China. So far, the complicated terms of anatomy have been finished and accepted by the Government as official. The terms in bacteriology, chemistry, and physics have also been finished and are awaiting recognition.

It is generally acknowledged that until the poorer classes practise hygiene, as well as the educated classes, infectious and other communicating diseases will not be stamped out. Knowledge of simple laws of health are, therefore, essential among high and low. With a view to carrying out such work, the Joint Council of Public Health Education was formed in 1916, consisting of representatives from the Y.M.C.A., National Medical Association and Medical Missionary Association, with offices at 5 Quinsan Gardens, Shanghai. The two secretaries are Drs. W. W. Peter and S. M. Woo. Their method of work consists in preparing travelling exhibits of automatic models, plans, lantern slides, movies and other methods by which the attention of the audience may be arrested and retained. Besides the above hundreds of thousands of pamphlets and booklets have been issued and sold at cost price. Among the titles of these books may be found: How to live, First Aid in the Home, Sanitation of a Chinese City, Tuberculosis, Infant Hygiene, Plague, Smallpox, Venereal Disease, Care of Teeth, Care of Eyes, Cholera, Sexual Hygiene for Young Men, etc. These books have had an enormous sale, showing that the public are interested in promoting hygiene.

Pressing needs in the Medical Line. Acting on the principle that "Discontent is the essence of progress," I am still far from satisfied with the speed at which sanitary matters have been undertaken by the authorities. The public health of the country should be invested in the hands of a business-like Central Health Bureau, with power to control epidemic diseases, and distribute vital statistics, manufacture vaccines, and perform analysis. In addition, a Central Medical Council should be established in Peking, consisting of leading medical men as well as representatives of the Government for the control and supervision of medical education and practice. The time has come when the old-style methods of practice should be absorbed into the modern methods, or else prohibited entirely in a limited number of years. The present system of simultaneously employing native-trained and western-trained medical officers in government hospitals and in the army is wasteful

and backward. Hygiene should be more widely taught in elementary and high schools, and properly trained school doctors employed to supervise the health of the pupils, so that all their defects, if any, may be at once discovered and corrected instead of being allowed to undermine the health and often shorten the lives of the children. The supervision of public health should be in the hands of proper sanitary boards, comprising leading physicians and business men as well as the police authorities. Social leagues should be founded wherever possible by leading citizens, men and women, and future mothers taught practical details for the management of a healthy home and family. The present tendency among the people, especially women, to buy patent medicines for headache, constipation, menstrual pain, backache, sleeplessness, and even sterility, is widespread, and can only be stopped by a fuller understanding of the laws of nature and a higher conception of the ethics of the medical profession. It may be further promoted by access to such public institutions as natural history museums, public health exhibits, industrial museums, commercial and technical displays, throughout every large city in the land. As the trade of the country expands, and technical and commercial education becomes more popular, these institutions will no doubt be established.

EARLY DAYS OF WESTERN MEDICINE IN CHINA

BY WU LIEN-TEH

Chinese Medicine has a long glorious history of its own, extending back to the days of Emperor Shen Nung (B. C. 2737-2705), patron deity of agriculture and medical science in this country, but the purpose of my address to-day is not to delve into that rather mysterious and complicated subject but merely to trace as far as possible the early connection of ancient Cathay with western (or modern) methods of treating sickness. In order to do this, we must ascertain the occasions when East and West first met in China and see if any exchange of medical ideas took place at such meetings.

So far as we know, it was during the reign of Wu Ti (B. C. 140-87), greatest of the Han sovereigns, when the Chinese Empire first extended its domains westward, that the earliest Euro-Asiatic contact took place, and this with the Romans largely through Turkestan to Ctesiphon on the Tigris, to Palmyra, the Persian Gulf and Gulf of Suez thence to Alexandria and Rome. Horace, Virgil, Ptolemy, Pliny the Elder and other writers have left their records behind on behalf of the west while Szu-ma Ch'ien (B. C. 145-74), the great historian of the Han Dynasty, was mainly responsible for our knowledge of Chinese chronological history up to B. C. 104.

Buddhism was officially introduced into China about A. D. 67 at the time of the later Hans, though a golden image of Buddha was supposed to have been brought back from Turkestan about B. C. 123, Anyhow, it was the Eastern Han Emperor Ming Ti (A. D. 58-76) who openly encouraged the new cult, which, originating in India, has had a profound influence upon Chinese culture and perhaps medical and scientific thought. The T'ang Dynasty came and went, medicinal springs where the beautiful Yang Kuei-fei drank and bathed were described in the old capital of Chang-an, the exciting game of polo was imported from Persia, and ideas of alchemy were exchanged with Arabia, but little or no authentic information is available as to the practice of western medicine having been introduced by foreign visitors or returned travellers.

The first European physicians working in China were probably scattered among the learned foreigners on the staff of the Mongol conquerors. In fact, it is stated that Aisie (perhaps

Isaiah) one of these *fuhllins* (or Frank) was courtphysician and astrologer to Kublai Khan and established a charitable hospital named Broad Charity in Peking in A.D. 1272. However, information handed down in regard to him is so vague that he appears almost as a legendary figure. There is also reason to believe that some medical activity was displayed by the Nestorian Christians, whose first churches were built in Honan as far back as A. D. 635¹. Later we read of the Nestorian Mar *Sergius* or *Sargis*, a physician from Samarkand, who in 1277 or 1278 was appointed governor of Chinkiang and built monasteries in and near this city, where probably medical aid was given to the poor.

China was visited in the latter part of the thirteenth century by Roman Catholic missionaries, and in 1307 the Franciscan, John of Montecorvino, reached Cambulac (Peking) and was appointed Archbishop by Pope Clement V.

The closing of the overland route to China led to a break in missionary endeavours to reach the country, until the sea route became better known. Francis Xavier attempted to reach China, but died in 1552 on an island off the coast of Kwangtung. From that date, however, China has been visited by a constant stream of Roman Catholic missionaries, particularly Jesuits. Their scientific knowledge soon won them the favour and esteem of the Chinese. Two of their number became Presidents of the Board of Mathematics (controlling the observatories and calendar affairs) in Peking.

Western practitioners had probably been working in the Portuguese Colony of Macao since its foundation. We know definitely that in A. D. 1569 the Misericordia Hospital (*Santa Caza da Misericordia*) was founded by Bishop D. Belchior Carneiro². Here medical aid was confined to Europeans only, as the relations between Chinese and foreigners in those days were not in the nature of friendly intercourse. About the same time, however, a start was made in providing medical relief by the Jesuit Fathers. Among the converts of the great Missionary Matteo Ricci (1552-1610) was the minister Hsu Kuangchi (Ko Lau) who received baptism under the name of Paul. His youngest daughter, called *Candida* in missionary annals, showed the same religious piety as her father³. Her history was quite remarkable. Married at sixteen years she became a widow at thirty. Thereafter she devoted her whole life to religious works, specially in the provinces of Kiangsi. Hu-kuang and Szechwen, whither she followed her son Basilius, Intendant General of the Posts and Navigation. Among the charities instituted by her in those parts was a foundling hospital and orphanage. As Du

1 A. C. Moule (Christians in China before 1550) 1930.

2 J. C. Thomson, Chin. Recorder, Vol. XIX.

3 Du Halde, General History of China, etc., London 1791.

Halde says, "the number of these children was so great that, notwithstanding all the care taken, upwards of two hundred died every year."

Soon afterwards the ranks of the Jesuit Fathers in China were joined by a great scholar and physician, Father Jean Terrenz (or *Terrentius*), called Schreck before he took holy orders. Born in A. D. 1576 at Constance in Switzerland, he became widely known and appreciated as physician, philosopher and mathematician. Skilful and successful cures at home endeared him to royalty and princes, who conferred upon him exceptional favours⁴. Yet at the age of 35 he renounced all splendours and joined the Jesuit Order. Being attached to the Overseas Missions he embarked from Lisbon together with another Father in April, 1618. Arrived in the East he undertook long journeys in India, Malacca, Sumatra, Cochin-china and China, everywhere collecting samples of minerals, plants and animals as well as undertaking climatological and ethnological studies. Being an excellent painter he supplemented his collections with creations of his brush, with the intention of embodying all in a bulky volume called "*Plinius Indicus*." Besides his manifold activities he found time to practice medicine and to convert patients cured by his skill.

Arriving in Macao in 1621 this brilliant man was-incredible to relate—first sent to Hangchow to work as an ordinary missionary and was summoned to Peking only when his services were required for revising the calendar. He concentrated all his energies upon this task which was not only of the greatest importance for the Chinese Government but at the same time provided the *raison d'être* for the presence of Jesuits in China. Whilst still in the preparatory work he died A. D. 1630. His *magnum opus* "*Plinius Indicus*" was unfinished at the time of his death and never published. Among less important works which appeared during his life time, one on structure of the human body published in the Chinese language deserves attention. Though later critics have, with some reason, dealt harshly with this small treatise (being poor both in text and illustrations) the historical importance of this first attempt to bring western medical knowledge to Chinese scholars should not be underrated.

Mention may be made in passing of Father Michael Boym (1612-1659) who in his turn tried to bring some knowledge of Chinese medical lore to the west by writing the *Clavis medica ad Chinarum Doctrinam de Pulsibus*. This comprised (a) four books by Wang-Chu-ho on the pulse, (b) a treatise on the aspect of the tongue in different diseases and (c) an exposition on simple drugs, prepared by the missionaries according to the directions of Chinese authors. The whole manuscript together with a

⁴ Pfister, Notices, Biogr. and Bibliogr. sur les membres de la Soc. de Jesus en Chine.

few other fragments was sent in 1658 by Father Couplet to Batavia to be dispatched thence to Europe. But due to disagreement between the Jesuits and the Dutch Company the author's name was suppressed and the book was published in 1682 by Andreas Cleyer, *Protomedicus* to the Company, under the title of "Specimen Medicinae Sinicae." Even some learned compilers of our times consider this as the authentic edition, although a book bearing the name of the right author and under its original title was published in 1686.

Still more interesting than the achievement of Boym were those of other Jesuit Fathers who, though not properly qualified medically, had an opportunity to prove to the court at Peking the superior qualities of certain western medicines. The famous Emperor K'ang Hsi (1655-1723) was attacked in 1692 by a malignant fever which was relieved by Fathers Gerbillon and Pereyra administering some medicinal lozenges prepared for Louis XIV of France. Later when recurring symptoms of Tertian ague appeared and defied the skill of the imperial physicians, proclamations were issued that anyone knowing of a remedy against this ailment should at once impart it to the Court where a special Commission would test it. The missionaries possessed a pound of cinchona bark which had been received by Father de Fonteney from India. They offered this and three patients confined in the Palace for experimental treatment were speedily cured by its action. Encouraged by this the emperor partook of the remedy with the same spectacular result.

Soon after his recovery, K'ang Hsi rode fearlessly from the palace into the city with a great following, and permitted the people, who as a rule were driven away when ever the emperor appeared in town, to remain in the streets, an event which had never happened before. Among those accompanying His Majesty were the four Fathers, Gerbillon, Bouvet, Fonteney and Vissdelou: they were allowed to stand while even the highest officials went down on their knees and touched the ground with their foreheads. In a loud voice, the emperor turning towards the missionaries said: "You Europeans have always served me with zeal and affection, and I have not the least thing for which to reproach you. Many Chinese mistrust you, but I, who have carefully watched your movements, am so convinced of your honesty and probity that I openly and publicly say: "You shall be believed and trusted!" K'ang Hsi then proceeded to tell the people how ill he had been and how the foreign guests had restored him to health again. A huge and commodious house within the First Court of the Palace was presented to the Jesuits.

We have noted that Terrentius had already compiled a book on human anatomy in Chinese. A more elaborate attempt in this direction was made by Father Dominique Parrenin (1669-

1741) who translated "L'anatomie de l'homme suivant la circulation du sang, et les nouvelles decouvertes par Dienis," into the Manchu (Mandarin) dialect. To the eight volumes of this translation the author added a ninth dealing with Chemistry, Toxicology and Pharmacology. After five years' labour this task was finished and he submitted the work to the aged Emperor. At least two handwritten copies of this work are extant.⁵

It stands to reason that the charitable acts instituted by Candida were continued and enlarged by successive missionaries, specially the homes for foundlings in whose souls rather than their bodily welfare the Church was interested. It is certain that about A. D. 1700 an organisation of lay Christians existed which provided among other things for the care of the sick. Besides, we know of quite a number of qualified medical men in the ranks of the Order who performed signal services for the indigent ones. Mention must first be made of Brother Bernard Rhodes (1645-1715), who arrived in China in 1699. Being able to benefit several patients who had been treated in vain by the native practitioners he soon won general confidence and opened a kind of dispensary for the poor in his house. Even the Court officials were impressed by this foreign doctor who contrary to the native practitioners, "talked little, promised little, yet performed much." The emperor himself was twice relieved by Brother Rhodes, once for palpitation of the heart, the second time for a boil on the upper lip. The treatment of the latter ailment necessitated the removal of a few hairs from the scanty beard of the emperor—a delicate operation which was entrusted to one of his eunuchs. Great was the ire of the emperor when he detected that *four* of the precious hairs had been clipped whereas *three* would have been *sufficient!*⁶ It would seem that Brother Rhodes gradually became regular medical attendant to the emperor whom he accompanied on his travels. To reward the Order for these services the emperor handed over gold ingots, then worth 200,000 francs.

Later Brother Joseph da Costa (1679-1747) worked from A. D. 1715 onwards in Peking where he kept a regular dispensary maintained by the gifts of paying patients. The work, was continued by Brother Etienne Rousset (1689-1758), from 1719. He accompanied the emperor Kang Hsi during his last journeys as physician and apothecary. Brother Rousset was generally known under the name of the Charitable Physician, and large crowds attended his dispensary. Another much loved physician was Brother Emmanuel de Mattos (1725-1764) who arrived in Peking in 1751. He was an able surgeon and, though urged by his superiors to become a full priest, he preferred to remain

⁵ One in Dudgeon Library, one in Russian Legation, Peking.

⁶ Dudgeon, Chinese Recorder, Vol. III.

a brother so as to have more time to care for the sick poor. Through constant overwork he contracted lung tuberculosis and died in 1764. The last of this series of physicians was Brother Louis Bazin (1712-1774) who spent the remaining six years of his life in Peking after a romantic career in Persia and India.

Reviewing these early medical activities of the Jesuits, it must be stated that though full of early promise they left but few permanent traces. The reasons are not far to seek. It would seem that the Superiors of the Order did not realise the great importance which medical work might play in propagating the faith or, because they possessed a strong foothold in Peking through their mathematical and astronomical skill, they paid insufficient heed to medical matters. Terrentius, for instance, with his splendid medical training, was first sent to Hangchow and only called back to the capital for consultation upon the calendar. Brother de Mattos was urged to become a full priest, and thus curtail his usefulness as a surgeon. Nevertheless, it is possible that the Jesuit Order would have continued to send out medical men to China if their whole work had not been cut short by the dissolution of the Society of Jesus in 1773. More than a quarter of century elapsed before a fresh attempt to introduce European methods into China was made by the British doctors in Canton and Macao. Before discussing this epoch in Chinese medicine it may be well first to cast a glance upon the medical activities of the Russian Ecclesiastical Mission in Peking.

Most of the regular missions dispatched from Russia possessed a medical man on their staff. The first was Pulart, a graduate of the Moscow Academy, who arrived with the Archimandrite Hilarion in 1716. In the same year there arrived also in Peking a *British* surgeon engaged by the Russians. The story is that the Emperor Kang-Hsi wrote to the Tobolsk Governor to recommend him a good physician and also some *serviceable physic for pleasure* (apparently vitalizer or aphrodisiac). A British surgeon of the St. Petersburg Hospital, called Thomas Garwin in the Russian records (his real name was probably Harwin), was selected. Together with some engineers sent by the Czar, this English doctor reached Peking in November 1716. All that we can find of his medical activity there is that he was once permitted to feel the pulse of the emperor. By the spring of 1717 he had already left the capital. Dr. John Bell of Antermomy (sic), the author of the curious work, "Travels from St. Petersburg in Russia to Diverse Parts of Asia" (Glasgow. 1763) also came to Peking with a Russian embassy. He arrived in November 1720, and left again in the following March. The Russian doctors seemed to have confined their attention to their own countrymen, the descendants of the valiant defenders of

the fortress Albazin on the Amur River who were brought to Peking by Chinese soldiers in 1688.

Allusion has been made already to the early existence of a European hospital in Macao. The Portuguese doctors who started it were gradually joined by Dutch physicians and later on by British medical officers. The first mention of a Dutch doctor is found in the year 1735, while in the chronicles of A. D. 1779 a Mr. Abraham Leslie, junior surgeon to the British factory, is mentioned. He was presumably an able man for he was recommended for promotion to the post of senior surgeon, but certainly he was smart in business as well, lending his savings at high interest to the Chinese merchants. On one occasion when one of his debtors got bankrupt, the doctor forcibly seized his house, disobeying the orders of his superiors to return to the factory. He proceeded similarly on a second occasion, though, as the chronicler drily remarks "he had certainly received more than the original principal." Leslie made himself so obnoxious that he was finally arrested by the Council in 1781. In 1783 he visited India where he was bold enough to complain personally to the Governor-General who, however, refused to support his claim.

Since Leslie's time one or two doctors had been permanently employed by the East India Company at Canton and Macao (between which two places the staff alternated, Macao being chosen for the off-seasons). Allusion can rarely be found of any medical aid given by them or their Dutch colleagues to the Chinese. In fact, the only occasion they treated such patients seems to have been when Chinese were wounded by Europeans. This is easy to understand since the Chinese laws of that period held anyone responsible for the death of a person to be guilty of murder and liable to execution. The foreigners therefore took a vital interest in the recovery of any Chinese who had been wounded by one of them. Apart from these comparatively rare occasions, the foreign doctors like the rest of their compatriots seem to have lived and worked quite separately from the Chinese. The desire to introduce Jenner's method of vaccination for smallpox into China finally bridged this chasm. The earliest attempts in this direction date back to June, 1803, when a dispatch was received from the Governor-General in Council of the East India Company in which he intimated his wish to see this method, which had come into general use in the British possessions in India, applied in China as well. The Governor-General therefore advised the Committee to consult the principal mandarins. Meanwhile, a supply of vaccine, sent by the Bombay Governor, was actually received in October 1803. With the aid of the hong merchants it was tried upon a number of healthy Chinese children but without success, "the virus having from the length of passage, been deprived of its virtue." Fortunately another

method of obtaining an active virus was effected. To describe this event we had best quote the words of Dr. Alexander Pearson, surgeon to the British factory, who unlike his Portuguese colleagues made the method available to rich and poor alike and was principally, if not solely, responsible for its gaining firm root among the Chinese population. In a report of 1816, Dr. Pearson wrote:

“In the spring of 1805.....the vaccine was brought by Mr. Hewit, a Portuguese subject and a merchant of Macao, in his vessel, upon live subjects from Manila—His Catholic Majesty having had it conveyed by suitable means and under the care of professional men across the South American continent to his settlement in the Philippine Islands. I observe that one of them (D. F. X. Balmis) states himself to have introduced the practice in this country; but before his arrival in China it had been quite extensively conducted by the Portuguese Practitioners at Macao, as well as by myself among the inhabitants there and the Chinese, and the accompanying tract, drawn up by me, had been translated by Sir G. Staunton into Chinese and published several months previous to his arrival. As I deemed the inoculation on any subjects connected with the foreign society, or with the Settlement of Macao, nugatory towards an establishment of the practice in China, it was from the beginning conducted, first at some expense, by inoculations at stated periods among the natives, and of them necessarily the poorest classes, who dwelt crowded together in boats or otherwise.....”.

The beginning was naturally difficult but, Pearson continues, “the method soon sprang into favour among the Chinese, who though very conservative in their feelings, when once convinced of the benefit of any new method, take it up very readily, and great numbers were brought to be operated on during the period of the raging of small-pox in the course of the winter and spring months of 1805-1806.”

Pearson now needed more help than the casual attention given him by his compatriots in the factory, and from 1806 onwards he employed Chinese assistants, recruited from the employees of the factory. The most prominent of these was Yao Hochun, called A. Hequa by the foreigners and nicknamed Dr. Longhead on account of the extra-ordinary length of his head. It is said of him that in the 30 years of his activity he vaccinated one million of people. Moreover, he taught the art to his son and thus became the founder of a family of distinguished vaccinators.

Dr. Edward Jenner in England soon learnt of the success attained in China. In a little tract called “Results of

Vaccination", is a letter from one John Barrow, who transmits to Jenner a copy of Sir George Staunton's treatise and informs him of the results achieved, adding that "thus the English at length as well as the other Europeans have established their claim (which though last is not least) on the gratitude of the Chinese." The great discoverer of vaccination (Jenner) was evidently much impressed, because according to an entry in Farington's diary he remarked to the latter that the Chinese seemed much readier to resort to vaccination than the English people nearer home.

However, things in China did not progress as well as during the panicky days of 1805-06, when thousands were vaccinated at a time, the Chinese assistants of Dr. Pearson working both locally under his immediate supervision and in the country districts. However, as soon as small-pox ceased to be epidemic, "the evil and the remedy against it were equally forgotten." The preservation of the virus in those days, when only vaccination from human being to human being was practised, necessitated regular vaccination the whole year round, but this was not always possible in China. Twice it was necessary to reintroduce virus from the Philippines, and twice it was found alive in the rural areas though dead in Canton. Gradually, however, all difficulties were overcome. The principal hong merchants established a fund for the gratuitous vaccination of the poor at all times and a small present was given to parents bringing their children. By this means it was possible to perform vaccination every ninth day throughout the year. Gradually the art also spread to the adjacent provinces and several attempts (at first futile) were made to introduce it into the capital of Peking. Finally this became successful in 1828 through the good services of the prefect Tseng who had formerly served in the South. (Among the descendants of the Albazines in Peking vaccination was practised from 1820 by the doctors attached to the Russian Mission who had introduced it to Kiachta on the Russian-Chinese frontier as early as 1805).

By the time Pearson left China in 1832 his great work had already been firmly implanted.

In his final report Pearson highly commended Chinese vaccinators, particularly A. Hequa, whom he called "a man remarkably qualified for the business, by his accuracy of judgment, method and perseverance." In the 1843 report of the Medical Missionary Society was published this eulogy, "the name of Alexander Pearson will ever be associated with those who have proved benefactors to mankind." Sir Andrew J. Ljungstedt, the President of the Swedish Factory and author of the book "Macao and China," said in 1834, "Dr. Pearson will ever live

in the memories of the foreign residents in China, as an attentive and sympathising friend, as well as skilful physician."

A description of the steady penetration of vaccination into the rest of China is interesting but space does not allow even a brief sketch. Therefore I will now turn to the second stage on the road of modern Medicine in China, namely, the foundation, in the year 1820, of a dispensary for Chinese at Canton by John Livingstone, surgeon to the East India Company and the Rev. Robert Morrison, D. D. We possess but little information about the former, "the first person who systematically brought medical aid within reach of the Chinese". It would seem that he had been in China since 1808⁷, although Morse, the chronicler of the British Company, mentions him for the first time in 1812, when serving as Assistant Surgeon under Pearson—with an "annual share" of £1,000. In the lists for 1815 Livingstone figures already as surgeon though still drawing the same salary up to 1825. Soon afterwards he apparently returned to England on long leave, for in 1827 Thomas R. Colledge figures as Assistant Surgeon under Dr. Pearson. In 1829 Livingstone is reported to have died at sea while returning to China. The life history of Rev. Robert Morrison is better known. Born in North-umberland as the son of a religious family he offered his services in 1805 to the London Missionary Society (founded in 1795). In the next year he took up a short course of medical study for missionaries at St. Barts, London. At the same time he began the study of the Chinese language through the manuscripts at the British Museum, in which arduous labour he was helped by a Cantonese student, Yong Samtuk.

Morrison left for China in 1807. Because of the petty hostility of the East India Company to Protestant missions and missionaries he had to travel via America, which country he reached after having been 109 days at sea. He then took passage via Cape Horn and arrived at Canton on September 7, 1807 where with the aid of two Roman Catholic Chinese he energetically pursued his language studies. In 1809 he joined the East India Company, drawing £500. as interpreter, and the same sum as instructor in the Chinese language. Dr. Pearson was one of his pupils in 1812. For printing and circulating his translation of the Bible by stealth he was dismissed by the Company in 1815. In the next year he joined the Amherst Embassy as an interpreter and thus travelled to Peking. In 1823 he went to England, returning again to China in 1826, and died in 1834 at Canton.

As can be gathered from a valuable article "Historical Landmarks of Macao" by Thomson⁸ Livingstone's idea in

⁷ J. C. Thomson, Chinese Recorder, Vol. XVIII.

⁸ Chin. Recorder 1887.

founding the dispensary was not only to give aid to the poor sick but to find out whether the Chinese Pharmacopoea "might not supply something in addition to the means now possessed of lessening human suffering in the west." He therefore invited the co-operation of Dr. Morrison on account of the latter's great command of the language. A Chinese medical library consisting of upwards of 800 volumes was installed with a complete assortment of Chinese medicines. A respectable Chinese physician, Dr. Lee, was engaged; occasionally a herbalist attended to explain the properties of the various articles supplied by him. Livingstone and Morrison spent one to two hours every morning at the Institution to supervise and assist Dr. Lee, who seems to have been in actual charge. Some help was evidently also given by Dr. Pearson, for he wrote in 1821:

"I have also been able to give pretty constant attendance and have had an opportunity of observing the details of Chinese practice, in from about 10-15 cases daily."

Strange to say he spoke only of Morrison as the founder and regular consultant of the dispensary and did not mention his regular assistant Livingstone.

The success of the dispensary was evidently assured from the very beginning, as shown by a statement of Livingstone himself republished in the above-mentioned article by Thomson⁹.

"Already much good has been done, much suffering has been relieved (hundreds were treated), and upwards of 300 patients have made very grateful acknowledgment for renovated health..... Besides our commercial intercourse, which is not always helpful to friendly sentiments between man and man, we have hitherto had little or no opportunity of establishing with them those friendly reciprocations of beneficent acts which must ever constitute the firmest bonds of social intercourse. Such attempts as this seem calculated to produce speedily the best results..... I am certain we have in the short time in which the institution has existed, fully proved that we are both able and willing to do them much good; and that both they and we have much useful information to impart to each other."

I have not been able to establish definitely how long this institution was maintained. As has been said, Morrison left China on furlough in 1823, while Livingstone went away after 1825, destined never to return again to the field of his benevolent activities.

Their places remained empty for but a short time. In 1827 Dr. Thomas Richardson Colledge (known as the Chinaman's

⁹ This had appeared before in 1821 in the Indo-Chinese Gleaner.

Friend), an old Rugby boy and Assistant Surgeon to the East India Company, began medical work for the Chinese at Macao. A very sympathetic account of Colledge was published in 1834 by Sir A. Ljungstedt under the anonymous name of "A Philanthropist." Here praise is bestowed upon the East India Company whose liberal policy "allowed to their medical servants salaries so ample, that they were satisfied, and went about doing good" and a fitting tribute is paid to the doctors themselves who

"were not the formal practitioners of their profession; they entered into the chambers of the sick, carrying healing and balm to the mind as well as body; they were patient hearers of the often-told and long details of enervated, distressed and melancholy minds. . . ."

Sir A. Ljungstedt quotes copiously from the medical pioneer's own records, some of which are here given. In a statement written at Macao in 1832 Colledge says:

"In the year 1827, I determined to devote a large proportion of my time, and such medical skill as education and much attention to the duties of my profession had made my own, to the cure of so many poor Chinese sufferers of Macao and its vicinity as came in my way. My intention was to receive patients labouring under every species of sickness, but principally those afflicted with diseases of the eyes. . . ."

"During that year my own funds supplied the necessary outlay. . . . In 1828 many friends who had witnessed the success of my exertions in the preceding year, and had become aware of the expenses I had incurred, came forward to aid in the support of a more regular infirmary. . . ."

"Thus the hospital grew up upon my hands; confidence was established amongst a people who had been accustomed to consider foreigners as barbarians, incapable of viruous, almost of human feelings; and the number of my inmates was regulated only by the limits of my accomodations. Two small houses have been rented at Macao, capable of receiving about forty patients.

"The more opulent and respectable classes of Chinese have in the last three years added their names to the list of subscribers; and have, by giving the hospital the sanction of their support, much enlarged the circle of its usefulness. The E. I. Company has written of it in terms of approbation, and when applied to, has liberally supplied it with medicines."

He concludes that:

"Independently of the practical benefits conferred on suffering humanity, it is most desirable that the enlightened

nation to which I belong should be known in this country as possessing other characteristics than those attaching to us solely as merchants and adventurers. As charitably anxious to relieve the distress of our fellow-creatures, we may be remembered when the record of our other connections with China has passed away."

Many Chinese testimonials, some still extant, praise Colledge's good deeds, but perhaps the most fitting tribute to him was a painting by George Chinnery, an exiled Irish artist residing at Macao. This shows an elderly Chinese woman blind with cataract led by her son to Colledge for aid. The operation was successful; in fact, the patient was on the point of returning home when the painting was made.

Besides his clinic at Macao, Colledge assisted by Dr. Bradford, an American physician hailing from Philadelphia, opened in 1828 a free dispensary at Canton, where big crowds of sufferers were relieved. The work was later carried on by Bradford and Cox, Assistant Surgeon to the Company, and it appears that the institution existed until its closure preceding the first Anglo-Chinese war (1840).

The Ophthalmic Hospital at Macao shut its doors in 1832, when—owing to the departure of Dr. Pearson—Colledge found no more time to attend to it. During the five years of its existence, some 6,000 patients had been treated. Remarkable as this achievement is, it forms but a portion of the benefits conferred by Colledge. For, though he himself modestly deprecated it, his example as well as his reports and papers were instrumental in directing the attention of the missionary circles in America and Europe to the splendid opportunities for medical missionaries in China and led to the early dispatch of the first and greatest of them, the Rev. Dr. Peter Parker.

This great man, of whom it was truly said that "he opened the gates of China with a lancet when western cannon could not heave a single bar"¹⁰ was born in Massachusetts in the year 1804 and graduated at Yale both in theology and medicine. Appointed by the American Board of Commissioners for Foreign Missions he left New York in 1833 on board the *Marrison* and arrived at Canton on October 26, 1834. Soon afterwards (December) he left for Singapore to study the Chinese language. There he opened a dispensary for Chinese where more than one thousand patients were treated from January to August 1835.

Returning to Canton he opened on November 4 of the same year a hospital and dispensary in Factory No. 7, Fung-taihong, San-taulan Street, a site quite near the foreign factories. It was fortunate for the young institution that this building be-

¹⁰ Rev. Dr. Beadle, quoted by Thomson, *Chin. Rec.*, Vol. XIX.

longed to Howqua (his real name was probably Wu Tunyuen), the richest of the hong merchants and the only one who had with westerners other than strictly business relations. For he waived aside the rent which had been settled at \$500.00 per annum.

In Parker's first quarterly report, he mentioned that the building had on the second floor a large room, where 200 patients could be comfortably seated and prescribed for; in addition the house afforded shelter to at least forty in-patients. Though it was planned to admit cases of all kinds if they were "of peculiar interest and praise", it was resolved that a single class of patients would probably furnish more than enough work. Eye diseases were chosen, because they were most common and the native practitioners were usually helpless against them.

To characterise the exceptional success of Parker's undertaking from the very beginning, a record left by himself may be quoted:—

"..... It was after long effort that a place was found for a hospital, and when at length a suitable building was rented and previous notice had been given, on the first day no patients ventured to come, on the second day a solitary female afflicted with glaucoma came, the third day half a dozen, and soon they came in crowds. It is difficult to convey to a person who has not visited the scenes of the hospital a just idea of them. He needs to be present on a day for receiving new patients, and behold respectable women and children assembling at the doors the previous evening and sitting all night in the street, that they might be in time to obtain an early ticket for admission. He needs behold in the morning the long line of sedans, extending far in every direction; see the officers with their attendants, observe the dense mass in the room below, stand by during the examination and giving out tickets to the hall above, where they are prescribed for, urgent cases being admitted at once, while others are directed to come again at a specified time..... Great numbers of patients are thus relieved every day, exhibiting more and more the confidence placed in the physician.

The idea to run the institution as an ophthalmic hospital, which—as we have seen—was never quite strictly adhered to, was soon given up altogether, general cases and especially surgery being taken up in addition. Kerr summarises in his *History of the Canton Hospital*:

"The surgical operations called forth the wonder of all, and well they might, for the excision of tumours, the operation for cataract, the removal of stone from the bladder, were methods of relieving suffering which had never been

heard of; and indeed the astounding fact became known that surgical instruments and operations were unknown throughout the empire."

This statement undoubtedly is exaggerated but there is no doubt that new and hitherto unheard-of surgical methods were first tried in this real cradle of western medicine in China. Mention may be made of the first lithotomy operation in 1844 (stones of the bladder are uncommonly frequent in Canton), the first ether anaesthesia in 1847 and the first chloroform narcosis a year afterwards. That bold operations could be confidently undertaken even before these methods to relieve pain were introduced, was possible because of the courage and apparent lack of nerves of the Chinese patients, repeatedly commented upon by pioneers of those days. Another favourable factor was that wound infections, which made the contemporaneous surgical wards in Europe such haunts of horror, were practically unknown in the Canton Hospital.

The foreign merchants residing in Canton not only took great interest in Parker's work by contributing liberally to its modest needs (the expense for the first quarter was \$454.84) but also visited the hospital, and some actually assisted Dr. Parker in his operations. Notable among the latter was Mr. W. Jardine, who—though he had given up his original profession of ship surgeon to the East India Company—was ever ready to lend to Parker all the assistance he could. In spite of this and the ready help from his colleagues Parker soon felt the need of native assistance. In 1837 he began with a class of three promising youths to whom he gave in addition to practical training theoretical instruction—apparently in the English language. The best of these students was Kwan Ato, a nephew of the famous painter Lamqua (pupil of Chinnery). This man soon became most proficient in surgery so that many operations, even serious ones, were entrusted to his skilful hands. He continued to serve in the Canton Hospital under Drs. Parker and Kerr except during the wartime of 1856-58 when he enlisted as surgeon to the Imperial forces sent from Kwangtung to fight the rebels in Fukien. Here he narrowly escaped death when the rebels surrounded a city in which he had opened a military hospital. For these services and as the first western-trained surgeon to the Chinese military forces he was rewarded by a crystal button with the title of mandarin of the fifth rank. On restoration of peace he returned to the Canton Hospital as Dr. Kerr's senior assistant, and from 1866 taught Practical and Chinese Medicine at the newly opened medical school. The remaining period of his life was spent in lucrative private practice. Kwan Ato died in 1874 and after him came a succession of Kwans as doctors.

But we must return to the year 1836, when Drs. Colledge and Parker together with the Missionary Elijah C. Bridgman (founder of the Chinese Repository) made a joint appeal for the foundation of a Medical Missionary Society in China. This, the first Medical Missionary organisation in the world, was actually inaugurated at a meeting held under Mr. Jardine's chairmanship in the rooms of the Canton General Chamber of Commerce on February 21, 1838. Dr. Colledge (though absent) was elected President, Dr. Parker, W. Jardine, C. T. Lay and E. C. Bridgman Vice-Presidents, Dr. Alexander Anderson, the successor of Colledge as surgeon to the British Factory, Recording Secretary, while among the members we find not only the names of physicians but of many business men, some very prominent in after years. The only Chinese member for years was Howqua, ever a generous helper of Parker's work. The lofty program of the society provided for medical missionary work in China on as large a scale as possible adding that "while the Society's agents, who will be looked for from Missionary Boards in Christian lands, will ply their art they will educate young Chinese in it and reflex benefits will accrue to medical science from discoveries in China." The first general convention of the young Society was held in 1843.

The earliest practical step taken by the Society was to send Dr. Parker during the time in which the Canton hospital underwent extensive repairs, to Macao to carry on hospital work in a house purchased by them (July-October, 1838). Parker then returned to Canton to continue the work until June 1840, when the disturbed state of affairs in China stopped it.

The fateful year of 1839, when the troubles leading to the first Anglo-Chinese war became threatening, saw the arrival of two more medical missionaries destined to play most important parts in the introduction of western medicine.

The first of these was Dr. William Lockhart who was commissioned by the London Missionary Society and arrived in January, 1839. The Macao Hospital, left vacant since Parker's return to Canton, was placed under his care, but in September he was compelled to leave on account of the measures taken by the Chinese Government against Britishers. He retired temporarily to Batavia, studying the Chinese language under the Rev. Medhurst and practising medicine amongst natives and immigrant Chinese alike.

The second new missionary was also an Englishman. Benjamin Hobson, born in Northamptonshire in 1816 and a graduate of University College, London. In September 1840, he jointly with Dr. W. B. Diver took charge of the Macao Hospital, while Dr. Lockhart, who had reopened

the institution a few months earlier, left for Chusan. The occupation of this island by the British seemed to offer suitable opportunities for medical missionary work. His hopes were realised, for by February 1841, when the withdrawal of the troops made a longer residence impracticable, over 3,500 patients had been treated, including many non-surgical cases. In addition, he attended the members of the Chinese Coolie Corps from the Canton district which had been formed to serve as a service detachment to the British army.

Lockhart returned to Macao, where he remained until the end of the war. After the treaty of Nanking in 1842 he proceeded to Hongkong in the hope of being sent immediately to Chusan, but was retained till summer 1843. He supervised the building of the Medical Missionary Society's Hospital in that new British colony. This was placed under Hobson's care, the Macao hospital having been disposed off. In July 1843, Lockhart resumed work in Chusan, achieving the same success as before. It became clear, however, that the port of Shanghai, just opened to the foreigners, would afford much ampler facilities. Thus, after he had paid two visits to this city towards the end of 1843, he closed the Chusan establishment in January 1844, and the following February started the Society's operations at Shanghai. However, before considering these post-war activities we must see how Parker fared during the war.

During the hostilities of 1839 the Canton hospital was removed from the factory to Dr. Parker's own residence and then to the Canton dispensary, evacuated by the British doctors. Finally, on June 7, 1840, it was again closed by reason of the blockade of Canton by the British; on the closing day there was still an attendance of some 200.

Parker, with the approval of the Society, left early in July, not so much to recuperate his health as to preach the cause of medical work to England and America. His hope was to raise a permanent fund for maintenance and enlargement of the Society's operations, as well as the education of Chinese pupils. He took with him a collection of paintings of the more remarkable cases, showing both the condition of each malady and the appearance of the patient after cure, donated by his admiring friend, the painter Lamqua; these he afterwards presented to Guy's Hospital Museum. Suffice it to say that Parker's presentation of the cause met everywhere with encouraging success and even led to such permanent results as the founding of the great Edinburgh Medical Missionary Society.

The war ended, Dr. Parker, who had married in America a niece of the statesman Webster, again reached China and with his bride took up residence at Canton on November, 5, 1842.

This was in direct opposition to the old regulation "that neither women, guns, spears, nor arms of any kind can be brought to the Factories." Mrs. Parker was the first foreign lady to reside at Canton, living as a "lone woman without a single female companion for many months." Curious reference is made to her in a memorial presented to the throne by the Imperial Commissioner Kiyong, hereafter a patient of Parker:

Another point, it is the wont of the foreigners to make much of their women. Whenever their visitor is a person of distinction, the wife is sure to come out to receive him. In the case of the American Parker and the Frenchman Langrene (? Lagrene), for instance, both of these have brought their foreign wives with them, and when your slave has gone to their barbarian residences on business, these foreign women have suddenly appeared and saluted him. Your slave was confounded and ill at ease, while they, on the contrary,, were greatly delighted at the honor done them. The truth is, as this shows, that it is not possible to regulate the customs of the Western States by the ceremonial of China, and to break out in rebuke, while it would do nothing towards their enlightenment, might chance to give rise to suspicion and ill-feeling.

Dr. Parker reopened the hospital on November 21 in the former premises. Old Howqua, the landlord, demurred at first, remembering the trouble he had when an inquest was held upon a beggar who had died in the hospital. He soon gave in, however, and when asked about the rent he refused any, saying:—"My own heart likes this business too; if any repairs are necessary, just call on my compradore and he will see that they are attended to."

This grand old man died of enteritis on September 4, 1843.

The success of the Canton Hospital became greater than ever, sometimes about one thousand persons being present on a receiving day. Yet the great changes engendered by the war were bound to reflect themselves upon the Medical Missionary Society, which owed its existence to the foreign community resident formerly at Canton but now removed largely to Hongkong. The crisis came in the year 1845 when the Hongkong members demanded that meetings be held at their place and questioned the administration of a sum of \$5,000.00 collected by Parker mainly in America. The committee demanded control of this sum, whereas Parker wanted to use it at his own discretion, a claim which was afterwards endorsed by the American donors. The result was that two committees and societies functioned, each claiming to be the original society and each having for its President, Dr. Colledge, who lived in England. Attempts to

reconciliate the parties failed and the two societies continued to exist side by side until, as far as I could establish, the Hongkong Society quietly passed away. Colledge remained President of the Medical Society for 40 years, that is, until his death in 1879 at the age of 82.

It will be well to refer here to the further history of Parker. Having acted with Dr. Bridgman as joint secretary in the negotiations of the U.S. Treaty with China at Macao on July 3, 1844, and as interpreter at the exchange of treaties at Pun T'ong, Canton (Dec. 31, 1845) Parker was appointed U.S. Charge d'Affaires. In 1847 he severed his connection with the American Board of Missions, but continued his medical services until 1855 at the hospital and amongst the foreign community, so far as his diplomatic duties permitted. Naturally, more and more of the medical work devolved upon his able Chinese assistants. In the spring of 1855 Parker returned to the U.S., the charge of the hospital having been transferred to Dr. John Kerr who had arrived in 1854. Appointed U.S. Minister Plenipotentiary he once more visited China but left the country for good in 1857 and took up his residence at Washington. When Dr. Colledge died in 1879, Parker became his successor as President of the Medical Missionary Society, showing great interest in its welfare until his death on January 10, 1888, at the advanced age of 84 years.

We may return now to the discussion of the activities after the war. One favourable point of the treaties, concluded in the years 1842-1844 between China on one hand and the United States and France on the other, was the insertion of the so-called Toleration Clauses which allowed foreigners the right to build hospitals and schools as well as places of worship in the treaty ports. Thus we find that in this period not only the work in Canton was safely continued but that medical missionaries gained a firm footing in other cities formerly not accessible to them.

Mention may first be made of the old port of Amoy where trade with Spain and the Philippines had been carried on for several years. Here the work was started in 1842 by Dr. William Henry Cumming who opened a dispensary at Ku-lang-su, a small island adjoining the port. He was soon joined by Dr. J. C. Hepburn. Both had to retire after a few years' work on account of ill-health. Their successors did not fare better, while Dr. Ino. Carnegie, who took over the hospital in 1859, soon severed the connection with his mission to join a local firm of medical practitioners. There being no new medical missionary available, he agreed, however, to carry on the hospital work—a voluntary service which was continued by his successors. This was very fortunate, because it enabled Dr. (later Sir) Patrick Manson

who arrived in Amoy in 1871, to take charge of the hospital and start his epoch-making discoveries in tropical medicine.

The year after the opening of Amoy, work was started in Ningpo by Dr. Lockhart who during a visit in summer 1843 treated about two hundred patients. In fact, it appears that he was doubtful for some time whether to make this place or Shanghai his future permanent residence. As we have seen, he finally decided upon Shanghai, while the post at Ningpo was filled towards the end of 1843 by Dr. D. J. Macgowan (American Baptist Mission) who, being generously supported by the foreign community of Bengal, was able to open a small hospital in 1845. Two interesting features of medical relief were started here: (a) treatment of opium addicts (b) lectures on anatomy before the practitioners and students of the city with the aid of models from Paris, some plates and one skeleton. Considerable interest was evinced by the class, one of which became Dr. Macgowan's valued assistant.

We have seen already how Dr. Lockhart commenced regular work in Shanghai in February, 1844. This was first carried on in a Chinese house, situated apparently in a southern suburb of the city and rented by the Hongkong Medical Missionary Society. The institution was a great success from the start:—

“As soon as the hospital was opened”, writes Lockhart, “and its purpose became known, crowds of people came daily to the house, urgently, often boisterously, requesting to be attended to. The applicants were not only residents in Shanghai, but many came from Su-chau (Soochow), Sungkiang and other cities in the vicinity, and also from the island of Tsung-ming.”

In 1846 it became necessary to provide ampler and better accomodation for the *Chinese Hospital*, as the establishment was then called. No footing could be found within the city itself, so a piece of ground situated more than a mile outside the north gate had to be chosen. Here a building with a large hall for out-patients and commodious wards was erected, the total cost together with land being \$3,200.00. The property was vested in the hands of some British residents at Shanghai, conditionally that it be always used for the purpose of a hospital. About the same time, in accordance with a wish of the subscribers to the Medical Missionary Society, a local committee was formed consisting of Messrs. Dallas, Shaw, Beale and Dr. Lockhart.

The improved accomodation added much to the usefulness of the hospital. The best proof that it enjoyed the confidence of the people is:

‘That during the various attacks on Shanghai city in the uncertain times of the Triad, Taiping and other rebellions,

both sides agreed, though Dr. Lockhart's hospital was very inconveniently situated for the hostile forces in their attacks on the city, to steer as clear of it as they could and particularly to avoid damaging it with their artillery; and both sides found the hospital ready to accommodate, to its fullest capacity, their wounded and their sick."¹¹

Like most of the medical missionaries in those early days Lockhart did his best to train assistants, one of whom, called Chun-fu, showed unusual ability. Dr. Lockhart went on furlough in 1857, his place being taken temporarily by Dr. Hobson, who after a year had to go home too on account of ill-health. The medical work was then carried on by Chun-fu with the occasional assistance of local practitioners until in 1860 Dr. James Henderson arrived on behalf of the London Missionary Society to assume charge.

The next port we have to deal with is Foochow, opened to foreign commerce in 1844. It was not till six years later that the first medical missionary, the Rev. Wm. Welton, arrived (1850). Though many sufferers applied for relief, his work was at first hampered by the hostility of the literati who resented the fact that Welton had obtained a portion of a temple near their schools. The dispute was finally settled by Dr. Welton removing to another temple nearby. Here he continued his activity until 1856 when his health became so debilitated that he was obliged to seek rest first at Shanghai, then in England. He did not recover, and died in 1858. The regular medical work at Foochow came to a standstill until 1870 when Dr. and Mrs. Osgood arrived.

Reference has been made already to the construction of a hospital at Hongkong by Dr. Lockhart and its opening by Dr. Hobson (June 1, 1843). Like the other early institutions this soon prospered; the number of patients for the first two years was over 7,000, so that in 1845 extensions became necessary. Hobson from the first paid particular attention to the training of pupils. In his 1845 report he bestowed special praise upon one of them, Ahsam by name, whom he considered capable of running a hospital like himself. Hobson adds:—

"I am very anxious to see a medical school established in the immediate vicinity of this hospital in Hongkong. And from the facilities such a desirable and useful institution as this would give to China, I trust no efforts will be spared to carry this project into effect."

The failing health of Mrs. Hobson made it necessary for the doctor to accompany her home in autumn 1845, but she died during the voyage. Dr. Hobson returned in 1847. During

¹¹ Brayton Barff, *North China Herald* 1926.

his absence the hospital had been kept open, first by Dr. Alfred Tucker,* surgeon to the Naval Hospital Ship and the Colonial Surgeon Dr. Francis Dill; then after the untimely demise of these two merited physicians by Dr. A. Balfour.

Soon after Hobson had resumed his labours in 1847 it was intimated to him by the Directors of the London Missionary Society that he should go to Canton where no English Missionary had been stationed since the death of Dr. Morrison in 1834. Complying with this wish he succeeded in April, 1848, to obtain a house in the western suburb of Canton, at Kum-li-fau. The hospital established there became so popular that in 1854 it had to be moved to more spacious premises. Hobson was fortunate in having a most skilful helper in Ho Kingmun who performed all smaller operations and fulfilled the duties of a resident surgeon.

In 1854 and 1855, when fighting between the Imperialists and the members of the Triad Society took place, the institution was busy in caring for the wounded. The strain of this work proved too much for Dr. Hobson and he left for Shanghai in December 1854. Ho Kingmun, however, ably carried on with the assistance of Dr. Walter Dickson. He was afterwards rewarded by the government with a white crystal button, corresponding to the sixth rank. During the fight native physicians were employed by the government to care for the sick and wounded outside the hospital. Those with the militia were paid *according to the number of cures they effected*. Naturally they preferred to send all serious surgical cases to the hospital which cared in 1855 for nearly 30,000 patients, 10,000 of these being new ones.

Successful as Hobson's hospital practice was, its importance is surpassed by another undertaking which he had started at Kum-li-fau. This was the compiling of *Chinese textbooks on medical subjects*, being translations of well-known English textbooks. The first was an *Anatomy and Physiology*, followed by a volume dealing with the principles of *Physics*. Next came a work on the *Principles and Practice of Surgery*, another on *Midwifery and Diseases of Children* and finally a treatise on the *Practice of Medicine and Materia Medica* together with an English-Chinese Medical Vocabulary. The engravings illustrating the books (many from original drawings made by Hobson's friend and voluntary helper Dr. Dickson) were executed by Chinese artists in Canton.

The success of the publications was from the first assured. The greatest compliment to the author lay perhaps in the fact that they were several times republished by the Chinese, the

* It was largely due to Dr. Tucker's efforts that in 1845 a "China Medical and Chirurgical Society" was organised, of which institution he was the first president.

Anatomy and Physiology at the instance of the father of the Viceroy of Canton. Publications of the whole series were made in Japan as well. Hobson's books remained for many years the standard works in Chinese, and their influence not only upon the Chinese in touch with western medical men but upon the scholars in general cannot be overstated.

We come now to another crisis in the intercourse of China with the foreign powers, leading to the war waged with interruptions between the years 1856 and 1860 and resulting in the conclusion of treaties which reaffirmed among other things the right of foreigners to build or open hospitals and similar institutions in the treaty ports. This specific clause as well as the security afforded to foreigners by the various treaties gave a mighty impetus to their medical work. Before dwelling upon this, we must contemplate how far the war interfered with the organizations already existing. The military operations being mostly restricted to Canton and its environs, the work at other medical posts appears to have been carried on without much disturbance. In Canton, however, it was completely interrupted. The premises of the Missionary Hospital there which had served all purposes since the early days and had continued rent free by Howqua's heirs, were destroyed by fire on December 14, 1856, when the foreign factories were burned by the Chinese. The dispensary, where most of the medicines were kept, had received the same fate in October.

Hobson's hospital did not fare any better. It being situated in the vicinity of some large batteries in the western suburbs, the British Consul insisted upon evacuation in October, 1856. Scarcely anything was removed, as hopes for an early adjustment of difficulties were entertained. For some time the Chinese in the neighbourhood voluntarily stood watch over the premises and their contents. But their protection had to be gradually withdrawn and soon only the bare walls remained. Dr. Hobson himself removed to Shanghai in February 1857, where he carried through the press his work on Surgery. Towards the end of the same year, when Dr. Lockhart was compelled by domestic circumstances to return for a while to England, Hobson was in charge of the Shanghai Hospital for Chinese until a year later his own health necessitated a furlough.

The interruption of the work was not of long duration. Dr. Kerr reopened the Medical Missionary Society Hospital in 1858 in a Chinese building in Chang-sha street in the southern suburbs where it remained until removed in 1866 to its present site. The Kum-li-fau institution was also reopened in 1858 under Dr. Wong Fun, the first Chinese to have graduated in medicine abroad. Dr. Wong Fun (Wong Cheuk-hing) was a

native of Heong-san district in Kwangtung and was first a pupil in the school of the Morrison Education Society under Samuel R. Brown. Together with two other Chinese students (Yung Wing and Wong Shing) he accompanied his teacher to America and took a degree in literature. He then proceeded to Edinburgh where—supported by the benevolence of some foreign merchants at Hongkong he studied medicine from 1848-1853. He passed with honors, taking several prizes and, after graduation as the fifth of his class, he took up post-graduate work in pathology and anatomy. When a student he was already under the influence of the Edinburgh Medical Missionary Society. As a graduate he offered his services to the London Missionary Society and was sent to China. Arriving in 1857 he first opened a dispensary in Hongkong but next year removed to Canton. He was superintendent of the Kum-li-fau hospital for about two years only, resigning on account of a dispute over the fraudulent and commercial conduct of some converts. He returned to Canton where he soon became prosperous in private practice and was eventually appointed Customs Medical Officer. He contributed many valuable articles to the Customs Medical Reports and took, from 1866 onwards, a prominent part in the school work of the Missionary Society hospital, which he was in charge of during Dr. Kerr's furlough in 1867. Having amassed a large fortune Wong Fun died on October 12, 1878.

Wong Fun's place in the Kum-li-fau Hospital was successively occupied by Drs. Happer, Carmichael and Dods until the establishment was amalgamated with the Medical Missionary Society Hospital and thus came under Dr. Kerr's control (1865).

During and soon after the war some interesting medical activities connected with military operations were carried on. We have mentioned already the signal services rendered during the Triad and other rebellions by Lockhart's Shanghai hospital. Worthy tasks of a similar nature were undertaken by the Catholics as well. Their mission had already established a hospital in 1848 at Tong-kia-tou, Shanghai, to take care of the refugees driven from Kiang-nan Province through famine after a disastrous flood. This establishment was probably of a temporary nature. Medical work was certainly taken up again in the same place during the occupation of Shanghai by the "Small Swords" (1853-1855.) Like the Protestant Missionary Hospital the church and residence of Tong-kia-tou lay between the two combating parties. Father Lemaitre not only remained at his dangerous post but cared for the wounded soldiers in a small building which ordinarily served as a Customs house (October, 1853). Brother Saguez, a trained layman, was sent

from Zi-ka-wei to help him, but more skilled help became necessary. Such was obtained from the commander of the French man-of-war "Cassini" who gave permission for his surgeons Fallier and Hubac to work in the hospital. This was heartily responded to by Fallier especially. Among the patients cared for was a future Viceroy of Nanking who ever remained grateful to the Catholic missions. Aid was given to civilians as well as military; in fact, a report of May, 1854, says that equal numbers of both were cared for. When the siege was over, the work probably came to a standstill for about 10 years, after which Brother Bernard resuscitated it with much success, caring for thousands of patients until his death in 1867.

During the war the French General Montauban organized a military hospital next to the St. Joseph's Church of Yang-king-pang, Shanghai, which was kept open as long as there were French detachments in the city but had evidently ceased to exist before 1864. It does not seem that medical aid was given to civilians on any large scale on this occasion.

During the occupation of Tientsin in 1860, the British established a small hospital there for the treatment of the wounded and sick of their Chinese Coolie Detachment, the doors of which were open also to the general public. Though this was a temporary undertaking only, it is of historical importance insofar as Li Hungchang obtained here his first insight into the benefits of western medicine. He never forgot the lesson and in 1881 personally opened a hospital in Tientsin, known as the Viceroy's Hospital, and remained its generous patron.

An interesting episode of a military nature is the organisation of the medical work in Gordon's 'Ever-Victorious Army', a well-trained Chinese detachment under foreign officers, engaged in suppressing the Taiping revolution during the years 1862-64.

The medical service of the detachment was in the hands of Dr. A. Moffit, up to that time assistant-surgeon of H. M. 67th Regiment, assisted by two other medical officers. A stationary hospital and a field detachment were organised—the former at Sungkiang first and later at Quinsan. The attendants were supervised by a young Chinese scholar who soon became most expert especially as a compounder of medicines. The field establishment was organised on the lines still used in the British army medical service nowadays. Usually two large covered boats served as its base, and from here the wounded were brought to Quinsan in Chinese gun-boats. Partly on account of the well-organized work and partly because the firearms of the Taipings were not of a very destructive

nature, the results obtained were excellent. Colonel Gordon testified: ¹².

“That the confidence felt by all ranks of his forces in the surgical skill of the principal medical officer was of signal service in nerving their minds for any enterprise, however hazardous.”

In the ‘Lancet’ (Aug. 11, 1866) it was stated that:

‘It is impossible to over-estimate the good done by Dr. Moffit, not only to the force in which he served with so much distinction, but to the reputation of his profession and country.’

One of the important consequences of the second Anglo-Chinese war from a medical viewpoint was that western medical work in Peking, interrupted for almost a century after the last Jesuit Fathers had gone, could be taken up again. The pioneer here, as previously in Shanghai, was Dr. Wm. Lockhart, now a F.R.C.S., who arrived in the capital as Senior Physician to the British Legation on September 13, 1861. He saw a few patients at first while staying as a guest of Mr. Bruce, the British Minister. As soon as he managed to settle down (October 23) in some premises adjacent, to and rented from the British Legation, such crowds came that the total number had reached 22,144 by the end of 1862. He continued to work until 1864 when, relieved by Dr. J. Dudgeon, he took up private practice at Blackheath in England. He died in 1896, long survived by his widow. In 1865, when the Legation wanted the premises for their own needs, the hospital was removed to a Buddhist temple on the Great East Street, now known as Hatamen Street. This Hospital is now named after Lockhart and is the nucleus of the present Peking Union Medical College.

New hospital accomodation in Shanghai had been provided some years previously. The site and plant outside the North Gate were sold in 1861 and a new building erected on Shantung Road. Thus the “Chinese Hospital” founded by Lockhart in 1844 became from now onwards to be known under the name of the *Shantung Road Hospital*.

Soon afterwards new hospital premises were also procured for the foreigners at Shanghai who had hitherto been cared for in the *Shanghai Hospital*, from 1861-1864 under Dr. Henry W. Boone. The accomodation becoming insufficient, shares were sold for the establishment of a new hospital, some French priests, among them Father Lemaitre, (died 1863) taking a prominent part in the organisation. A big building was rented from the banker Yang Taki, situated on the French Bund at

¹² Andrew Wilson (The Ever Victorious Army), London 1868.

the corner of Rue Colbert. The establishment was opened on January 1, 1864, the nursing being placed in the hands of the French Sisters of St. Vincent de Paul. It was first known under the names of the *French Hospital*, its present designation of the *General Hospital* coming into common use only when it was removed in 1877 to its present site north of the Soochow Creek.

In 1866 another important undertaking was started. With a capital fund of M\$84.00 (!) and in a house the rent of which was M\$5.00 per month, a dispensary was opened by the Reverend (afterwards Archdeacon) Thomson and Rev. H. N. Woo, the medical service being undertaken by Dr. J. Macgowan. Soon many patients attended and an appeal was made in the newspapers for additional funds which resulted in the prompt subscription of Tls. 700.00. With this, 13 houses were purchased at a cost of ten taels each and on the land thus acquired a small hospital was built, known at first—on account of its location—as the *Hongkew Hospital*. Several physicians including Dr. Jamieson, volunteered their free services. In 1880 the institution moved to its present site donated by Li Chiuping and assumed the name of St. Luke's. In 1881 the Gutzlaff Hospital, which had existed in a back street for about 20 years, was amalgamated with it.

As after the first, so after the second war, advantage was taken of the opening of new treaty ports. Thus Dr. William Gauld began to work at Swatow in 1863, Dr. F. Porter Smith in 1864 at Hankow, while in 1864 Dr. James Maxwell reached Formosa after a voyage in a sailing vessel lasting nearly six months and opened a hospital at Takow. This was so greatly appreciated that

“Go wherever you will (not excluding some savage districts) “Ma I-seng” is spontaneously spoken of and kindly inquired after”¹³.

Dr. Maxwell removed in 1866 to Taiwanfu, his place at Takow being filled by Dr. (afterwards Sir) Patrick Manson, newly appointed Customs Medical Officer to the Port.

Here I may conveniently end my narrative. While it has been possible in the foregoing pages to furnish an outline of the main events connected with the early introduction of western medicine into China, a summary of what may be gathered between the lines of the contemporary records is now called for.

The hospitals of those days formed just a rough shelter for in-patients, who were expected to provide their own food, clothing and often bedding. No trained nurses were available, the

¹³ Myers, Customs Medical Reports 1881-82.

care of the sick being usually left in the hands of relatives and friends, though pioneers like Peter Parker did not hesitate to sit for nights at the bedside of some patients upon whom they had performed serious operations. Greatly to their credit, the doctors amidst their manifold duties found time to teach their assistants. But, as in the case of physicians in medieval Europe, their pupils were more like apprentices than proper medical students. It was rarely possible for the overworked clinicians to interest themselves in scientific or public health matters. Even if they did, there was at that time no medium in China for recording their observations except in the hospital records, which had to be written to attract lay subscribers at home rather than medical readers.

With the year 1860 came the dawn of a new era. The organisation of a properly paid Customs Medical Service by Mr. (later Sir) Robert Hart from the year 1863 resulted in the publication of a series of valuable Customs Medical Reports, to which the best practitioners of the time contributed. This and the establishment of a regular medical school attached to Canton Hospital (1866) are the harbingers of the third great chapter of modern medicine in China.

HOW I BUILT HOSPITALS IN CHINA

BY WU LIEN-TEH

I have often been asked to place on record an account of the method by which I have established the series of hospitals in various parts of the country, especially in view of troublous times through which China has passed during the last dozen years. Before doing this a few words of introduction may be necessary.

The year 1905 saw a great movement among all classes of Chinese to follow the progressive ways of the west. The Imperial authorities of Peking decided to adopt a Constitutional form of government and so despatched a Commission headed by Viceroy Tuan Fang with a staff of over fifty persons to different countries of the world to study the question. In Tientsin, Viceroy Yuan Shih Kai had accomplished splendid results in military, educational and commercial affairs through the assistance of keen, experienced, western-trained men, headed by Mr. Tong Shao Yi. The Imperial Army Medical College had just been started for the training of medical officers for his model army. I was then in the Straits Settlements having returned from England and Europe three years previously. Thanks to the recommendation of my old friend, Admiral Cheng Pi Kwang (who was later on—1917—assassinated by the agents of militarists in Canton), I was asked to come up to the north and assist in the organisation. I decided, however, to take a flying trip to England and Germany and study at first hand the methods adopted by the Royal Army Medical Corps in London and also by the General Staff in Berlin. Here I stayed for six months, and then returned to China in October 1908, only to learn that Viceroy Yuan Shih Kai had received Irish promotion to Peking as one of the Grand Councillors, and that both the Empress Dowager and the Emperor Kwang Hsu had recently died within a few hours of each other. Instead of my would-be patron Yuan Shih Kai, I found the powerful General Tieh Liang, his great rival (who died in 1920) installed at the War Office. Fortunately, his right-hand man was Admiral Tan Hsueh Heng, who as junior officer to my uncle, Captain Lin Kuo-chang, was in England during the years 1896-99 superintending the construction of Chinese warships at British shipyards. Through his efforts, I obtained an interview with General Tieh Liang and also my appointment as

Vice-Director of the Imperial Army Medical College. Whatever one may say against the dilatoriness or incompetence of Manchus, one has to confess that no finer or more courteous types of *gentlemen* existed in China, and such men as Tieh Liang, Yin Chang, Prince Chun (the Prince Regent, 1908-11), Hsi Liang (Viceroy of Manchuria, 1908-11), could not be equalled anywhere in the world for their kindness to strangers and juniors.

I assumed my new duties as Vice-Director of the Medical College, then situated at Tientsin, in November 1908, my immediate superior being Dr. Hsu Hua-ching. The teachers were mostly Japanese, who lectured in their language either direct or through interpreters. But the greatest handicap to success lay in the absence of a proper hospital, where the students could be taught clinical work. The only institution available was a small hospital situated in the centre of the city with a few beds, ill kept and poorly managed. Hence during my three years at Tientsin, I made frequent trips to Peking and interviewed successive Ministers and Vice-Ministers of War to plead for the establishment of a modern well-equipped hospital, where not only could students be efficiently trained, but soldiers could be properly cared for and the graduate medical officers continue their practice, so as to be ready for all emergencies.

But my words fell on deaf ears, and though plenty of money was found for uniforms and ammunition, none could be spared for a hospital. As a last resort, I asked for the construction of a model hospital to care for the twenty thousand odd Imperial troops stationed in the capital, but even this was not granted. One of the excuses was that our Chinese soldiers, like our Chinese people, did not care for western medicine, but my seniors forgot that in time of war native treatment would be of no use, and the few western-trained men in the army without regular practice could not cope with emergencies. Up to this day, in spite of revolutions and successive interprovincial strifes, no military hospital worthy of the name is as yet established in the capital and as a result mission hospitals or charitable hospitals like Dr. Gray's and the Union Medical College are frequented by men in uniform for ailments which should be treated by their own medical attendants.

It was not until the great Manchurian Plague of 1910-11 and its aftermath—the International Plague Conference held in Mukden under my Chairmanship in April 1911—that an opportunity came for the realisation of my ideals. Among the resolutions passed by that Conference of eleven nations, were the following:—

13. The need for isolation of pneumonic plague patients being urgent, permanent isolation hospitals should be available. Such isolation hospitals should admit of individual isolation, be of rat-proof construction, and be capable of easy disinfection.
42. A permanent sanitary nucleus should be formed, capable of rapid expansion in time of plague, and a list should be drawn up of medical officers who could be sent immediately to the affected area on the outbreak of plague.
44. With the view of giving effect to these recommendations, every effort should be made to organise a central public health department, more especially with regard to the management and notification of future outbreaks of infectious diseases.

At this point it may be necessary to classify the hospitals for whose construction I have been responsible into:

1. Those, where the initiative has come from me, e.g., Harbin and other Plague Prevention Hospitals.
2. Those initiated by local provincial authority; North Eastern (Mukden), Tsitsikar Hosp.
3. Those initiated by a non-governmental body and organised by me, e.g., Peking Central Hosp.

1. Plague Prevention Hospitals, Manchuria.

The establishment in 1912 of the Manchurian Plague Prevention Service with headquarters at Harbin was a serious attempt made by the Chinese Government to give effect to the recommendations of the Mukden Conference mentioned above. Its inauguration was somewhat delayed by the Revolution, which started in October of the previous year, but fortunately the Viceroy of Manchuria (Chao Erh-sun), the Inspector-General of Customs (Sir Francis Aglen), the Vice-Minister of Foreign Affairs (Dr. W. W. Yen) and the late Commissioner of Customs (Mr. W. Haines Watson) all took a keen interest in the matter and did what they could to promote its success. For instance, the Viceroy appropriated from the Manchurian revenue Tls. 50,000 for the Hospital at Harbin, Tls. 40,000 for Manchouli, Tls. 30,000 for Tsitsikar and Tls. 20,000 for Lahasusu; Sir Francis Aglen induced the Diplomatic Body of Peking, which at first vetoed the scheme, to change their minds and to agree to the withdrawal of Tls. 60,000 annually from the Chinese Maritime Customs for the maintenance of the Service; Dr. W. W. Yen showed his sympathy by drawing up some of the regulations and placing the Plague Prevention

Service under the *aegis* of the Wai Chiao Pu (Foreign Office): lastly, Mr. W. Haines Watson (late Commissioner of Customs, Harbin, died 1914) who was present during the great plague of 1910-11 and therefore fully understood the urgent need of preventive measures, gave invaluable advice in the organisation of the several hospitals at the beginning of their existence.

Owing to the prevailing impression among western people that Chinese executives, however able or experienced, could not be trusted with money, it was agreed to appoint in addition to the Director and Chief Medical Officer, a Lay Director and Treasurer in the person of the Harbin Commissioner of Customs, "who will be responsible for the safe-keeping of the funds allotted for the maintenance of the Service, keep the accounts, issue salaries and supply the necessary funds for all expenditure duly sanctioned by the Director and Chief Medical Officer." On the other hand, in order to keep the Service efficient without undue interference from a layman, "the Director and Chief Medical Officer has general management of the Service, including the appointment, distribution and dismissal of the technical staff; he has also the power at his discretion to authorise appropriations to be made for any special purpose so long as that purpose is connected with plague and medical work and investigations, and provided the yearly limit of Tls. 60,000 is not exceeded." This division of labour has been found to work on the whole satisfactorily during the last twelve years, although one or two misunderstandings did arise through different interpretations of the regulations by succeeding Commissioners. A few words may now be devoted to each hospital.

(a) *Harbin*. The extensive plot of land, covering four English acres, on which the present Hospital stands, was once disputed territory claimed by the Russian Railway Administration. But, when we defined the object of our Service, General Horwath (then Head of the C. E. Railway Administration) gladly parted with it in 1911, and we now possess full title-deeds to this valuable property. The central office of our administration was at first situated in the Customs House, but as this arrangement was inconvenient to the Customs staff and ourselves alike, we moved to a rented building for two years, after which we occupied new quarters within our spacious hospital. Since the completion of our administration block in 1920, the office has been located here. The Harbin Hospital occupies two separate compounds, the west containing buildings devoted to administration and quarantine for 400 persons, and the east containing isolation blocks for the accomodation of 30 suspects and 40 plague cases. This Hospital

has been described in detail in the 1911-13 Report, and only the new additions need therefore be mentioned here. The original cost was \$70,000. Owing to the fact that the foundations of the several buildings were laid down towards the end of 1911, when the cold weather had more or less set in, it was soon found out that the walls showed a tendency to crack and the floors to rise in winter. Repairs were constantly necessary, and a few months after the World War was declared and when prices were rapidly rising, I suggested to the then Commissioner of Customs (Mr. Grevedon) to utilise some of the money we had saved during the past years in constructing a new double storey steam-heated hospital, but he demurred because of the uncertainties of the political situation. The opportunity passed, and before long the rouble (currency then prevailing throughout North Manchuria) rapidly declined in value until it reached practically zero-point, and as a consequence our hard-earned savings were totally lost. A fresh beginning was now made, and from October 1917 onwards our annual appropriations were paid in Chinese silver dollars instead of Russian currency. But both the staff and exchequer of the Service had suffered terribly. With the arrival of a new Commissioner of Customs (Mr. R. C. d'Anjou) in 1919, it was decided to build a new block, which would accomodate the plague research laboratory, museum, library and offices. In view of our limited funds, since no special appropriation was granted by the Government, the greatest economy had to be practised. We bought our own raw materials in winter and allowed no waste. The result was that we managed to complete a fairly large building having a wide foundation over seven feet deep with steam-heat and modern plumbing throughout for under Mex. \$18,000. This structure did yeoman service when Pneumonic Plague broke out in Harbin in January 1921, for, besides caring for 3125 plague patients (all fatal) from January to May, we were able to undertake extensive researches. These have been mostly described in the 1918-22 Report. One point, however, needs recording. On February 19th, 1921, Dr. Yuan Teh Mao, who had been on duty as Chief House-to-House Inspection Officer, showed symptoms of plague infection. I was at the time away at Suifenhö (about 360 miles east) and returned to Harbin on the evening of the 20th. Owing to insufficient accomodation, Dr. Yuan and I had been occupying two adjoining rooms next to the Plague Laboratory on the first floor of the new building. His illness necessitated my removing to a poorly heated room in one of the barracks, where I was joined the next day (that is, the date of Dr. Yuan's death) by Dr. J. W. H. Chun, Dr. Pollitzer and Dr. Young (a missionary colleague on a visit to us). Our cultures numbering

some hundreds of tubes also occupied the same bedroom as ourselves during the disinfection of the new block, and we passed through some anxious nights. Dr. Yuan died early on Feb. 21st. This incident shows the danger to which our staff is exposed when insufficient funds are forthcoming for the accomodation of a medical staff who have to spend day and night within the precincts of a plague hospital in time of epidemic. Sixteen months previously (1919), when Cholera raged throughout Manchuria, we admitted 1962 seriously ill patients with only 275 deaths (i.e. 14.11%).

In 1922, another new block costing \$30,000 was added to the Harbin Hospital for the accomodation of general patients. This building contains two first class wards, two second class wards and four third class wards, totalling 45 beds. There is also a fine operating room as well as rooms for X Rays and photography. In May of this year (1924) foundations were dug for the erection of an up-to-date Laboratory for Plague, Serum and Diagnostic investigations, costing \$25,000. When this is completed we shall have separate new buildings for (a) Administration and Medical Officers Quarters, (b) General Hospital and (c) Laboratory, Library and Museum, and thus avoid further makeshift arrangements.

(b.) *Sansing (Ilan)*. This is a town of about 15,000 inhabitants situated on the Sungari River, 150 miles north-east of Harbin. Owing to its being a port of call for river steamers, one of our hospitals was established there. For this purpose, some ready made buildings, numbering six large blocks within a fenced compound of $\frac{3}{4}$ of an acre, were bought in 1913, and reconstructed so as to accomodate 60 persons. The cost approximated \$10,000.

(c.) *Lahasusu (Tung Chiang)*. This small village of 1,000 people is situated at the junction of Rivers Sungari and Amur, and therefore occupies a strategic position. Although no plague had ever occurred here, it was considered necessary, in view of its importance as a port of call, to construct a modern isolation hospital in the neighborhood. Government land, measuring $1\frac{1}{2}$ acres, was purchased at a nominal price, and new buildings costing \$20,000 were erected. These consist of a two storeyed brick building for the Medical Officer above and Out-patient Department below, as well as separate Quarantine and Observation Blocks. There is accomodation for 42 patients. During the troubles following the Russian Revolution, the "White" leader Kaminoff and his followers surrendered to the Chinese troops and were later on sent to Kirin. It was due to his attempt to escape from prison and to obtain concealment in the local Russian Consulate that brought on the closure of

all Russian consulates in China and terminated the recognition of the Russian Minister, Prince Koudacheff, in Peking.

(d) *Taheiho (Helampo or Sakhalen)*. This is a growing city on the southern bank of River Amur and faces the Russian town of Blagovestchensk on the north. It is six days by steamer from Harbin. The Tsarist Government used to trace every infectious disease among man and beast, in Russian territory, e.g. anthrax, plague, swine fever, etc., to this locality, although it was, and is, one of the healthiest of places.

A fine brick hospital, functioning for normal and epidemic times, was built in 1913 and officially opened in 1914. It contains a large two storey block, as well as four quarantine and infectious blocks, accomodating 70 persons. The total cost was \$28,000. Our hospital has been the scene of many encounters between 'Whites' and Bolsheviks, both of whom applied for medical treatment and protection. The Japanese Military Red Cross also received our hospitality, and many Japanese wounded were treated within our walls during their fights with the Bolsheviks in 1920-21.

(e) *Manchouli (Lin Ching)*. This is the boundary town of Manchuria on the west adjoining Siberia. It has ever been the gate of entry for Pneumonic Plague from the endemic centres of Transbaikalia, and hence a hospital for observation and research at this place is absolutely necessary. In 1911 the Viceroy of Manchuria gave Tls. 40,000 for building a proper hospital at Manchouli. Foundations were forthwith dug in the spring of 1912, and much building material was purchased, but during the succeeding Revolution, the Mongols fanned by Russian expansionists burnt our place and left nothing behind but the bare ground. Several years then passed, and it was not until the second Manchurian Plague epidemic of 1921 came that we were able to borrow some buildings from the Municipality for our preventive work. Since that time we have stationed a permanent sanitary staff at Manchouli, sending expeditions into Siberia whenever occasion demands it and cooperating with our Russian colleagues in the happiest way. In 1923, we bought a ready built stone house for \$9,000 for the purposes of a research laboratory and quarters for our medical officer, at the same time retaining a large wooden block lent by the Municipality as Polyclinic. At this station is situated the large apparatus for disinfecting tarabagan skins with formalin gas before export. At present there is an embargo upon export and hunting by the Heilungkiang Governor, otherwise millions of skins could be disinfected and exported with benefit to all concerned.

(f). *Newchwang*. The Quarantine Hospital of Newchwang is the latest addition to the Manchurian Plague Prevention Service. For several years the need of such a hospital had been realised by all classes of the community because of constant invasions of Cholera from Shanghai, Japan and Korea. But it was not until 1918 that the appropriation was finally sanctioned. Building operations were commenced in 1919, and the hospital was formally opened on July 10th, 1920. There is a large front block 162 feet wide containing operation and diagnostic rooms and general wards; next is a disinfection block and behind lies the contagious block with individual rooms and verandahs facing the south. The cost of these original buildings is Tls. 40,000 and accomodation is for 45 beds. In 1923 a series of six detention blocks, built of bricks and having cement floors, were added to the hospital. Each block possesses a set of hygienic *kangs*, insect-proof and dirt-proof, to serve as beds for those detained under observation. There is accomodation for 80 persons in each block, and hence a minimum of 400 persons may be detained at any one time. The cost of this second lot of buildings is \$30,000. The total ground covers 3½ acres and lies on the south bank of the River Liao.

2. *Those initiated by local provincial authority.*

(g). *North-Eastern Hospital, Mukden*. One of the results of the internecine war between General Chang Tso Lin on one hand and General Wu Pei Fu on the other in 1922 was the awakening of the military leaders to the need of providing qualified surgeons for the hundreds, nay thousands, of soldiers wounded by modern fighting weapons. Both sides at the time had to rely mainly upon foreign doctors and institutions for the care of their wounded. Hence, in addition to the employment of younger, energetic Chinese to replace the older opium-sodden officers, General Chang and his son decided to construct a model military hospital in Mukden, partly to treat their soldiers and partly to train medical officers. For this purpose they entrusted me with its planning and organisation. A brilliant Chinese architect, S. S. Kwan, trained in America, was employed to draw the plans, the condition being that the hospital could be used, whenever necessary, for the public as well as military. The verandah system was chosen, with a central two-storey block for administration and officers ward, and individual unit wards radiating from a central pathway. Each ward possesses a complete heating and plumbing plant as well as rooms for nurse, clinical diagnosis, service, and lavatories. Altogether 26 out of 35 blocks have so far been built. Besides wards, there are three operating rooms, all equipped in the most up-to-date manner, separate departments for venereal diseases, skin, otolaryngology, eyes, and accidents. A deep artesian well has been dug, from

which pure water is pumped with an engine to a high tower. There are platforms to which the railway track leads. The total cost is over \$600,000. including \$140,000. for plumbing and heating. There is accomodation for 400 beds. This North-Eastern Hospital is easily the model military hospital in China, although many defects are still present. It is hoped that an efficient medical staff may be employed to run the hospital which will be opened this summer (1924).

(h). *Tsitsikar (Pukuei)*. This city, the capital of Heilungkiang Province, was given Tls. 30,000. by the Viceroy of Manchuria for the establishment of an anti-plague hospital in 1911 as a result of the great epidemic. The local Taoyin, Sung-Siao-lien (later on promoted to Governor of the Province) requested me to help in planning and equipping the Hospital, which was formally opened in 1912. There is accomodation for 60 patients. The Medical Officers are not directly appointed by me, though my assistance has been frequently sought in times of epidemic. The running expenses are provided by the local authorities.

3. *Those initiated by a non-governmental body and organised by me.*

(i). *Peking Central Hospital*. This is the institution to which I devoted my best efforts continuously for four years, because it was intended to make it the model *civil* hospital of China. The scheme originated with a private visit made to Mr. Chou Hsueh Hsi, (Minister of Finance during President Yuan Shih Kai's term in 1915) who was desirous of establishing a sanatorium costing \$100,000. at the Western Hills, where he used to spend regular week-ends. I pointed out to him the more urgent need of an up-to-date general hospital in the capital, which could serve as a model for both officials and people, and thus promote the interests of scientific medicine. The sanatorium could then be built on a smaller scale as an adjunct for convalescents. The upshot was a meeting of influential Peking residents in the spring of 1915 at the Central Park Committee Room. Among those present were Messrs. Chou Hsueh Hsi (Minister of Finance), Chu Chi Chien (Minister of Interior,) Tsao Ju Lin (Minister of Foreign Affairs), Chang Chung Hsiang (Minister of Justice), Lin Chang Min (Chief Secretary of the Cabinet), C. C. Wang (Councillor, Ministry of Communications), Sze Sao Tseng (Director-General, Lung Hai Railways), Chief of Police Wu, myself and a dozen others. \$110,000. were promised at the meeting besides the \$100,000 at the disposal of Mr. Chou. A fine plot of government land in the west city next to the historical Temple of Imperial Ancestors was presented to the Hospital, and another acre situated behind was afterwards

bought for \$13,000. to complete the site. An American firm of architects was engaged to draw up suitable plans, the building contract was given to a German, and I was appointed Honorary Medical Director to supervise everything except the finance. Foundations were dug in June 1916. The uncertainties all over the country brought about by the Japanese 21 Demands and Yuan Shih Kai's attempt to become Emperor considerably hindered the campaign to procure funds. This meant more intensive efforts on the part of the organisers, and greater need for economy in every direction. For instance, the Government Railways charged half rates for all materials conveyed by them; the Maritime Customs and Peking Octroi allowed free import to our goods; steel rods for the concrete work were supplied by the Hanyang Ironworks at 20 percent discount; the Chee Hsin Cement Co. deducted 15 percent from their regular prices; Mr. Sze placed his accountants at our disposal, and neither Treasurer nor Medical Director received any salary. Even the British firm of Twyford & Co., (with Mr. Thomas at the head) offered their services as our Tientsin agents without any commission. Everywhere I went I solicited subscriptions and travelled as far as the South Seas to interest wealthy friends who subscribed \$30,000. I myself donated \$2,500. Through a personal appeal to Mr. Liang Chi Chiao (Minister of Finance 1916), he granted a further \$30,000 from the Ministry as well as an annual allowance of \$1,000. for upkeep. Valuable medical and surgical instruments were also presented by Admiral Sah Chen Ping, then in charge of the Arsenal at Tehchow. Thanks to such hearty co-operation from all sides we were able to build and equip a thoroughly modern ferro-concrete \$400,000 hospital for only \$300,000. There is accomodation for 10 first class, 20 second class and 120 third class patients. A full account of this hospital appeared in the *Modern Hospital* (America), April 1917.

The new Hospital was formally opened on Jan. 27th, 1918, the whole of Peking turning up for three days to inspect it. But an unfortunate dissension soon occurred between the Treasurer, Mr. Sze, and myself. We had been working harmoniously for three years during the formative period, and no person could have been more liberal or keener on the success of the institution. At the completion of the building, however, he was advised by interested persons to create for himself the post of Hospital-lord (院董) with power to control the actions of the Medical Director instead of cooperating with him as an equal, as he had hitherto done. Many friends intervened to mend the rift, but there was already another doctor waiting to replace me on my departure. I resigned a short time after the opening, and was thus prevented from realising my ambition to *run* as well as *build* the hospital as a model institution, where our Chinese medical men and

women, rapidly increasing in numbers, could find a joint meeting place for their professional activities. Nearly six years have now passed since its establishment, and this great hospital, to which so many friends devoted their time and money, is being managed, as so many government institutions in Peking are, without vigour, enthusiasm or efficiency, although ample funds have been provided for maintenance and improvement.

Other Activities. Besides the above permanent hospitals, I was successful in inducing the Central Government in 1911 to build a series of quarantine sheds at Mukden, Koupangtzu, Shanhaikuan, and Newchwang as precautionary measures against the plague. These were all erected in haste during the winter months, of corrugated iron sheetings without wooden floors. The cost of each varied from \$20,000 to \$40,000 according to capacity. The minimum accomodation was 1,000. and the maximum 3,000. All these have now tumbled or been torn down except those at Newchwang and Shanhaikuan. When the Rockefeller Medical Mission visited China in 1915, I was invited by Drs. Welch, Flexner and Peabody and Mr. Roger Greene of the Commission to express my opinions as to the advisability of establishing a Medical College and Hospital in Peking and also to the kind of language in which medicine should be taught. The unrivalled Peking Union Medical College and Hospital, opened in 1921, are now included among the sights of the capital. In 1917, I petitioned the Foreign Office and Ministry of Finance regarding the need of establishing a Central Hygienic Laboratory in Peking with which our Manchurian Plague Prevention Service could be affiliated. The dispatch sanctioning my petition with the necessary appropriation had actually been received at the Foreign Office from the Ministry of Finance, but the next day it was withdrawn. Fortunately, after the outbreak of Pneumonic Plague in Shansi (1917-18) the surplus left from the one million dollar loan from international bankers was utilised for the building and equipment of this Hygienic Laboratory in the historic grounds of the Temple of Heaven. This Institute is now turning out useful vaccines and sera and receiving an annual grant of \$110,000. from the Maritime Customs. There have never been more numerous or more capable Chinese medical graduates in China than at this present moment, and it is hoped that every one will regard it as his bounden duty to push forward the interests of his humane profession for the promotion of public health as well as the mere routine treatment of the sick. Apart from public hospitals, private ones are essential, but these should be properly equipped with up-to-date instruments for diagnosis and treatment, not necessarily in a lavish or wasteful manner. Only by this means can Chinese hope to win their conservative people over to a just appreciation of the benefits of modern medicine.

SUMMARY OF PERMANENT HOSPITALS ESTABLISHED

No.	Location	Year	Accommodation
1 a	Harbin (i)	1912	470.
	„ (ii)	1920	Administ. and Lab.
	„ (iii)	1922	Gen. H. 45.
	„ (vi)	1924	Lab. and Museum.
b	Sansing	1913	60.
c	Lahasusu	1912	42.
d	Taheiho	1913	70.
e	Manchouli (i)	1921	30.
	„ (ii)	1923	Lab.
f	Newchwang (i)	1920	Isol. and Gen. 45.
	„ (ii)	1923	Quar. 400.
2 g	Mukden (N. Eastern) ..	1924	Gen. and Milit. 400.
h	Tsitsikar	1912	Gen. 60.
3 i	Peking (Central)	1918	Gen. 150.

ABSTRACT OF NOSOLOGICAL ARTICLES

BY J. W. H. CHUN

1. *The Differential Leucocytic Count in Beri-beri.*

Manchurian Plague Prevention Service
Reports, Vol. II, p. 61.

II. *Remarks on the Incidence of Certain Diseases in Chinese
and Europeans*

Manchurian Plague Prevention Service
Reports, Vol. IV, p. 199

III. *The Influence of the Chinese Diet on Diseases.*

Manchurian Plague Prevention Service
Reports, Vol. V, p. 283

IV. *On the Comparative Frequency of Non-Pulmonary Tuberculosis in North China.*

Manchurian Plague Prevention Service
Reports, Vol. VI, p. 281

I. *The Differential Leucocytic Count in Beri-beri.*

As very little attention has been paid to the blood in Beri-beri in text books, the author examined the blood of 42 cases of clinically recognised beri-beri in the Shantung Road Hospital, Shanghai, in 1912, and recorded the differential leucocytic counts.

The seasonal prevalence of the disease seemed to be in summer. Only males were examined, mostly young adults. Soldiers predominated in number. The acute "wet" type formed the largest number of the cases.

Table Showing the Differential Counts of:—

	Normal Chinese Standard. (author's previous Normal European standards publication Hutchinson in C.M.J. Vol. Schafer & Rainey XXIX No. 3)			Beri-beri
Finely Granular				
Oxyphile	60-70%	70%	58.0%	57.4%
Coarsely Granular				
Oxyphile	1-10%	2-4 %	5.7%	5.2%
Basophile				
Leucocytes	rare	0.5 %	0.6%	0.4%
Hyaline	5%	2-4 %	9.8%	19.5%
Lymphocyte	15-30%	22-25%	25.3%	17.2%

It may be noted that the hyaline cells averaged as high as 19.5% whereas the normal Chinese standard is only 9.8%. This noticeably high percentage of the hyaline cells was thought to be of help in the diagnosis of beri-beri in doubtful cases.

Since the hyaline cells also increase in other conditions such as malaria, care was exercised to exclude malarial subjects.

The author concluded by suggesting that as the etiology of beri-beri is obscure, the fact that the hyaline cells are relatively increased might act as a support to the theory that beri-beri is a disease caused by protozoon, because in other protozoal diseases such as malaria, kala-azar and trypanosomiasis, the percentage of the hyaline cells is augmented. This theory tends to support the statement of Castellani and Chalmers who said in their text book on Tropical Diseases "to summarize from the evidence, it appears more likely that a parasite will be found to be the spreader of the disease (beri-beri), which makes it more probable that the actual cause will be found to be protozoon than that it is due to diet, which, however, may be a predisposing cause, especially if the nutritive value of the food is low, or the proportions wrong."

II. *Remarks on the Incidence of Certain Diseases in Chinese and Europeans.*

The author maintained there is a racial partiality for certain diseases and that there are diseases or features of a disease which

are frequently met with in the Chinese but which are rare in Europeans and Americans. For instance, why is appendicitis comparatively rare among the Chinese whereas fistula-in-ano is so common? As very little is known at present about some of these peculiarities, it was thought profitable to rouse discussion and to start investigation.

Some of the diseases found to be common among the Chinese are enumerated:—

a) *Fistula-in-ano.*

This is exceedingly common. The cause of this condition was thought to be due to habitual constipation and the coarse food, though other causes such as tuberculosis etc. also come into question. Libbey (1) stated that he saw large numbers of the disease in Wuhu. These cases form 16% of his operative cases (72 out of 455).

b) *Tuberculosis.*

Tuberculosis in all forms is wide-spread, often seen in the last stages. A table showing the percentage of the different forms seen during a period of 10 months in 1923 is as follows:—

Glands	748	43.8%
Skin	312	18.3%
Bones	257	18.0%
Lungs	196	10.9%
Joints	133	7.8%
	<hr/>	<hr/>
	1646	98.8%

c) *Malignant scarlet fever.*

Scarlet fever in Europe is not such a dangerous disease; the mortality may be said to be about 3%. In China, however, this disease was unknown previous to 1873. Consequently it exhibits a severe form in face of an apparent lack of immunity. In epidemics in 1923, some families in Harbin lost all their children. When the foreigner is infected in China, he is apt to develop a severe and sometimes fatal attack, the case fatality among 68 foreign patients admitted to the Shanghai Isolation Hospital from 1905 to 1916 was 15.4%.

d) *Syphilitic rheumatism.*

For ten months, 689 cases of syphilis were seen and the resultant diagnoses tabulated as follows:—

1st stage	125	18.00%
2nd „	489	70.97%
3rd „	75	10.88%
4th „	0	0 %
		<hr/>
		99.85%

The preponderance of the secondary stage is partly accounted for by the large number of syphilitic rheumatism cases. For instance, during 1922-1924 (four years), 1490 syphilitic rheumatism patients were seen, but not one case of locomotor ataxia or general paralysis of the insane.

The following are comparatively rare conditions:—

x) *Appendicitis.*

During the last four years, only two cases of appendicitis were admitted, and from all accounts from other hospitals, the Chinese do not suffer from it as frequently as the Europeans. In the Harvard Medical School Reports, admission figures are interesting, as both Chinese and Europeans were received into the hospital.

Appendicitis cases among Europeans—20 cases in 240 admissions 8.3%.

Appendicitis cases among Chinese—10 cases in 766 admissions 1.3%.

y) *Carcinoma.*

Carcinoma is not really so rare in China. From the appended table, it can be seen it is often met with. Numerous factors, however, prevent the surgeon from seeing or operating on the sufferer. Still, it seems rare when compared with European figures.

z) *Chloroform as an anæsthetic.*

The Chinese patient takes this anæsthetic very well. During eight years, only one death from chloroform was recorded. In the Shantung Road Hospital, Shanghai, there was one death in two years. Dr. So To Ming of the Canton Hospital administered chloroform to over 10,000 patients during a period of 3 decades without a death.

TABLE A.
Diseases Which Are Comparatively Common Among The Chinese

	Plague Prevention Service Hospitals 1918-1922 Admissions 1325		Shantung Road Hospital, Shanghai 1914-1922 Admissions 19219		Red Cross Hospital, Soochow 1917 Admissions 1898		Union Medical College, Peking 1916-1917 Admissions 1199		Temple Hill Hospital, Chefoo 1916 Admissions 332		Red Cross Hospital, Shanghai 1914-1917 Admissions 986		Diseases of China, Jeffreys and Maxwell		Shanghai Health Officer's Report May 1918
		%		%		%		%		%		%			
Fistula in ano	78	5.9	338	1.9	2	0.1	58	0.5	13	3.9	16	1.6	One of the commonest surgical Conditions		
So t Fibroma	15	1.1	26	0.1			4	0.3	0	0	2	0.2	Very common and large		
Low mortality in influenza	1 in 24	4					0 in 3	0							The mortality is likely to be very low
Low mortality in typhus	2 in 56	3.6											Mortality 10 to 20 %		
Low mortality in Small-pox	1 in 9	11.1													
Syphilitic Rheumatism	28	2.1													
Piles	26	1.9	119	0.6	20	1	23	1.9	14	4.2	8	0.8	Exceedingly Common		

TABLE B.
Diseases Which Are Comparatively Rare Among The Chinese

	Plague Prevention Hospital 1918-1922		Shantung Rd. Hospital, Shanghai 1914-1922		Red Cross Hospital Soochow 1917		Union Medical College, Peking 1916-1917		Temple Hill Hospital, Chefoo 1916		Red Cross Hospital, Shanghai 1914-1917		Diseases of China, Jeffreys & Maxwell		Principal of Medicine, W. Osler	
	Admissions 1325	%	Admissions 19219	%	Admissions 1898	%	Admissions 1199	%	Admissions 332	%	Admissions 986	%	1 in 2000	%	American figures	%
Appendicitis	2	0.15	29	0.1	27	1.4	11	0.9	1	0.3	10	0.98	1 in 2000	0.05		
Tabes dorsalis	0	0	0	0	0	0	0	0	0	0	0	0	0 in 12000			1.2
Carcinoma	7	0.53	41	0.2	10	0.5	27	2.3	8	2.4	15	1.5	External Common Internal rare		201 in 16562 cases	
Death from chloroform	0	0	none recorded	-	0	0	0	0	0	0			Exceedingly rare			
Sprue	0	0	0	0	0	0	0	0	0	0	0	0	Rare			
Varicocele	0	0	0	0	0	0	1	0.08	0	0	0	0	Rare			
Diabetes	0	(1) 0	0	0	0	0	0	0	0	0	2	0.2	Few recorded		276 in 27618 admissions	0.9
Rickets	0	0	0	0	0	0	0	0	0	0	0	0	Rare			50 % to 80 % of children
Rheumatic Fever	0	0	8	0.04	0	0	7	0.6	1	0.3	1	0.1	Rare		285 in 9286 admissions	
Hernia	12	0.09	73	0.38	9	0.5	16	1.3	2	0.6	8	0.8	Very Common			
Chorea	0	0	0	0	0	0	0	0	0	0	0	0	None			
Hysteria	0	0	4	0.02	0	0	3	0.3	0	0	0	0	None			
Pes Planus	0	0	1	0.005	0	0	2	0.2	0	0	0	0	Not mentioned			

(1) 85 Cases seen in 91,000 out-patients—0.09 %

III *The Influence of the Chinese Diet on Diseases.*

The author tried to establish that some common diseases among the Chinese may be due to diet, and reversely the absence of certain diseases may be accounted for by the nature of the food of the people.

When the food of the common people of North China is examined, it is found to be almost entirely vegetarian in nature and monotonous in the extreme. The small merchants and coolies eat boiled Kaoliang, some vegetables fried with soya bean oil and bean curd in the morning. In the evening they eat man-tou (bread made of wheat flour), vegetables in season, bean curd, and fun-tiao (vermicelli made with peas). On the 1st and 15th of each month and at festivals the merchant class eat meat in some form, while the labouring class rarely eat meat, fish or eggs. Cow's milk is never used of course. Because they eat twice or thrice daily and because the vegetarian diet is not sustaining, they have to take bulky meals.

Hutchison (Food and Dietetics p.180) calculates that a vegetarian should consume about 2960 grm of cooked food daily. But the total capacity of an ordinary stomach is only 1200 grm. Thus one can see it must be filled to the limit of its capacity at each meal.

Adolph (China Medical Journal 1923 p.1013) on investigating the food of the middle class of North China found that the fuel value per capita for 60 kg body weight amounts to 3355 calories of which 87% were furnished by cereals. The Occidental standard for 70 kg body weight for moderate muscular work is 3500 calories and for light work 3000 calories. The amount of protein ingested per capita is found to be 111 grms of which only 5.3 grms are animal in origin in strong contrast to the diet of the Occident where about 50% of the total is of animal origin. He further maintains that the food of the Chinese consists of 94% of "vegetables," and that in consequence almost all the protein, fat and carbohydrates are derived from them.

The author claimed that one may connect all these dietetic drawbacks with the prevalence of many diseases in China. For instance wide-spread tuberculosis may be due in part to under-feeding together with the absence of animal protein and the small amount of fat in the diet.

Then the strict vegetarian aliment tends to diminish energy both mental and physical, as well as the power of resistance to disease.

The bulkiness of the diet leads sooner or later to derangement of the stomach and bowels. During a period of two and a half years, there were 29,552 out-patient visits to the Harbin

Hospital and out of these, 2357 were for diseases of the digestive system.

The prevalence of fistula-in-ano and piles may be accounted for in part by the coarse and bulky food.

Large numbers of sufferers from beri-beri, and the occasional occurrence of scurvy and pellagra testify to some form of diet deficiency.

Turning to the other side of the question, we find that Rickets is almost unknown because of the universal breast-feeding of the children, and the absence of slum-conditions.

Diabetes is not rare among the well-to-do class, but it is not frequently met with among the poor.

Summarising from the records of the Manchurian Plague Prevention Hospitals, one may say the following diseases are comparatively infrequent:—appendicitis, gastric ulcer, gastric cancer, gout, rheumatism, gall stones, renal stones and obesity.

IV On the Comparative Frequency of Non-Pulmonary Tuberculosis in North China

After commenting on the prevalence of tuberculosis in China supported by mortality figures in Hongkong (311 per 100,000 living) in 1922, Shanghai (102, 108, 109 and 140 per 100,000) in 1923, 1924, 1925, 1926 respectively, the author tried to show that there is a preponderance of non-pulmonary forms in North China while the pulmonary form is more frequent in South China, and tropical areas such as British Malaya. The term non-pulmonary forms means, tubercular involvement of the lymphatic glands, bones, joints, skin, peritoneum, intestine etc.

In Malaya, Dr. J. Tertius Clarke (Malaya Medical Journal Vol II No. I March, 1927) and Dr. Nathan Raw found that non-pulmonary tuberculosis was exceedingly rare. In the Government Medical Report of the F.M.S. in Perak for 1925, under the heading "Tuberculosis" there were 2571 cases treated in the Government hospitals, but under "local diseases" of joints, bone, skin and glands, there was no mention of tubercle.

Leaving the tropics and examining cities further north in China, it is claimed that the pulmonary form is less frequent and that the non-pulmonary form is more so until in Harbin the former comprised only 18.1% of all forms of tuberculosis while the latter 81.75%.

The appended table and chart tend to support this contention.

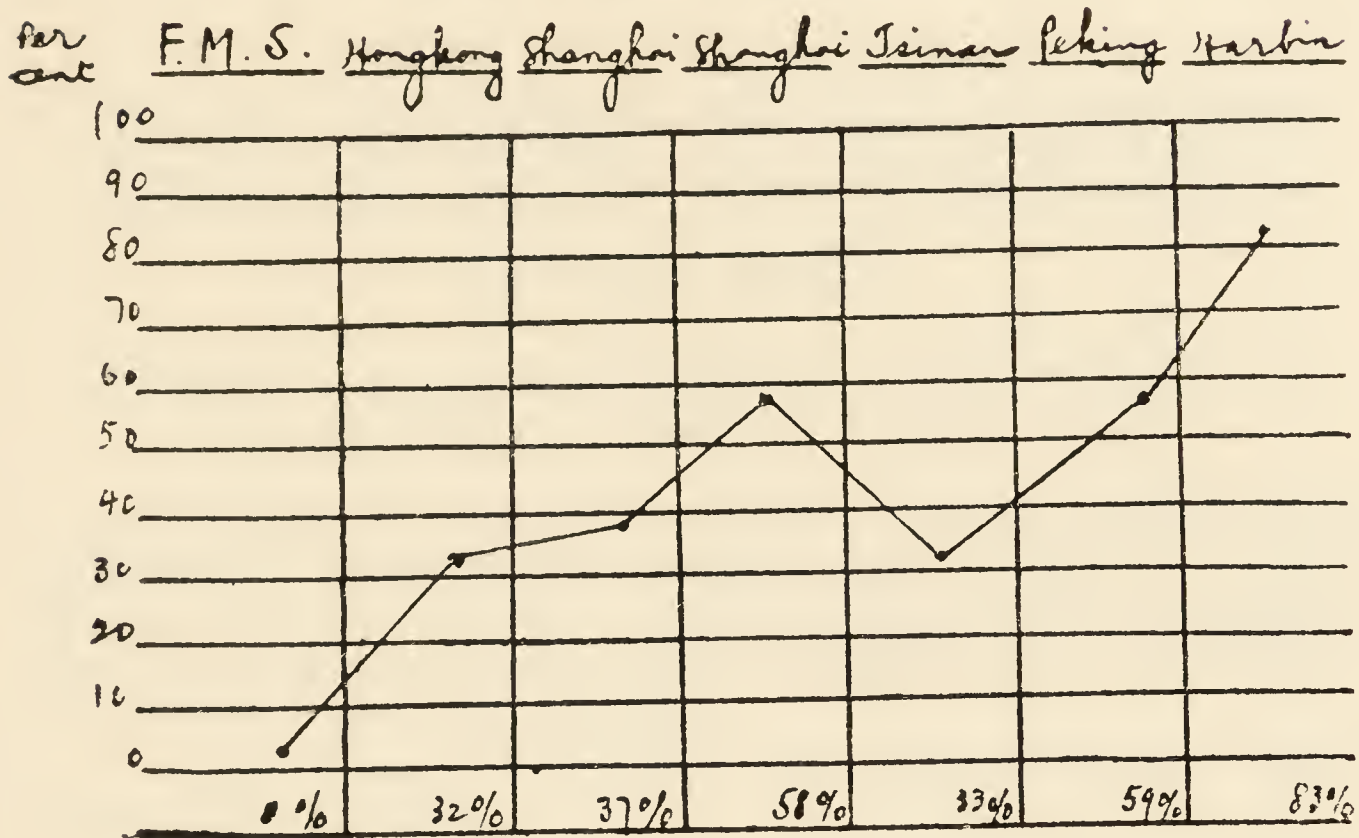
Conclusions.

1. 1. In the F.M.S. the non-pulmonary forms of tuberculosis seem to be exceedingly rare, unless imported, while the pulmonary form is very prevalent.
2. In South and Central China, the non-pulmonary forms seem to be more frequently seen, but still less in proportion to the pulmonary form.
3. In North China, the non-pulmonary forms seem to be very prevalent, while the pulmonary form is much less in proportion.
4. It is suggested that the cold and dry climate in the North has an influence on the prevalence of the non-pulmonary forms, either per se, or together with the ill-ventilated and crowded conditions of housing obtained in those regions, and that the hot and damp climate in the tropics favours the pulmonary form of tuberculosis.

TABLE I
Showing Incidence Of Different Forms Of Tuberculosis

	Harbin Hospital Out-patients (1924-1927)		P. U. M. C. Out-patients 1916-1917		Shanghai Red Cross Out-patients 1917-1918		Tsinan Out-patients 1921-1922		Shantung Road Hospital In-patients 1917, 1919, 1922, 1924, 1925	
	No.	%	No.	%	No.	%	No.	%	No.	%
Lung	538	18.1	117	41.2	80	63.5	469	66.5	189	42.6
Bone	620	20.9	50	17.6	6	4.7	85	12.0	74	16.7
Skin	327	11.1	0		0		0		0	
Abdomen	28	0.95	9	3.2	4	3.1	44	6.2		
Glands	1,065	35.9	84	29.3	20	16.0	105	14.8	64	14.4
Testis	41	1.4	0		0		0		0	
Joints	296	9.9	24	8.4	16	12.7	3	0.4	116	25.9
Elsewhere	47	1.6	0		0		0		0	
	2,962	99.85	284	99.7	126	100.00	706	99.9	443	99.6

Percentage Of Non-Pulmonary Tuberculosis Traced
From F.M.S. To Harbin



BLOOD GROUPING IN NORTH-EASTERN MONGOLIA AND IN NORTH MANCHURIA

BY H. M. JETTMAR

(Summary of an article appearing in the "*Mitteilungen der Anthropologischen Gesellschaft in Wien*," 60, 39)

Investigations were made regarding the blood groups of the population of North-eastern Mongolia and of North Manchuria during a stay in Urga and a journey through North Manchuria. This territory is inhabited by three different tribes: 1. Mongols; 2. Northern Chinese; 3. Manchus.

(1) Among the Mongols the percentage of the four blood groups was as follows:—

Total cases (166) O=26.6%, A=27.7%, B=30.7%, AB=15.1%. Index=0.93.

Pure Mongols (114) O=28.6%, A=23.2%, B=31.3%, AB=16.9%. Index=0.83.

(2) Among the Northern Chinese (668 cases):

O=28.1%, A=29.5%, B=33.2%, AB=9.1%. Index=0.85.

Probably the resemblance of the index of the Mongols and the Northern Chinese may be due to the fact that in the past centuries these tribes have mixed promiscuously.

(3) Among the Manchu only the Goldi could be investigated. The percentage is as follows:

O=39.3%, A=18.9%, B=36.2%, AB=5.6%. Index=0.585.

LIST OF WILD RODENTS KNOWN OR SUSPECTED
TO SUFFER FROM PLAGUE

By WU LIEN-TEH and R. POLLITZER

I. TABLE OF RODENTS (OTHER THAN THE DOMESTIC RATS AND
MICE) DEFINITELY KNOWN TO SUFFER FROM SPONTANEOUS PLAGUE

Revised to December, 1932

	Name:	Locality:	Reference:
SCIURIDAE	1. Tarabagan <i>Arctomys bobac</i>	Transbaikalia and Mongolia	Bjeliavski & Rjeshetnikoff, Vj. Obst. Guig., 1895, 26, No. 4.
	2. Marmot <i>Arctomys caudatus</i>	Southern Kirghisia	Kalina, Vj. Microb. & Epid., 1930, 9, 549.
	3. Dauria sisel <i>Spermophilus dauricus</i>	Transbaikalia	Skorodumoff, Studies upon the Epid. of Pl. in Transbaikalia & Mongolia, 1928, 39.
	4. Large suslik <i>Citellus fulvus</i>	South-East Russia	Koltzov, Vrach. Gaz., 1917, 147.
	5. Small suslik <i>Citellus pygmaeus</i>	South-East Russia	Deminski, quot. by Klod- nitzki, Russki Vrach, 1913, 1067 & Berdnikov, Ztrbl. f. Bakt., 1913 69, 251.
	6. Ground squirrel <i>Geosciurus capensis</i>	South Africa	Mitchell, S. Afric. Publ. H. Rep. f. Year end. June 30, 1924.
	7. Californian ground squirrel <i>Citellus beechyi</i>	California	Wherry, Jl. Inf. Dis., 1908, 5, 485 & McCoy, U. S. Publ. H. Rep., 1908, 23. 1289.
	8. Squirrel <i>Fonambulus palmarum</i>	Ceylon	Hirst, Colombo Health Rep., 1922, 17, 41.
	9. Squirrel <i>Sciurus palmarum</i>	India	Simond, Ann. Inst. Pas- teur, 1898, 12. 664.
MURIDAE Murinae	10. Multimammate mouse <i>Rattus coucha</i>	South Africa	Mitchell, Jl. Hyg., 1921, 20, 377 & Haydon, Lancet, 1921, 2, 1103.
		South-West Africa	Fourie, S. Afric. Publ. H. Rep. f. Year end. June 30, 1932, 71.
		Uganda	Uganda Protect. Ann. Med. & San. Rep. for 1921, 96.
		Belgian Congo	Schwetz, Fornara & Col- lart, Ann. Soc. Belge de Med. Trop., 1929. 9, 219.
		Senegal	Leger & Baury, Bull. Soc. Path. Exot., 1923, 16, 136.
	11. Striped mouse <i>Rhabdomys pumilio</i>	South Africa	Mitchell, Jl. Roy. Army Med. Corps, 1906, 6, 130 & Report 1924.
	12. White-tailed rat <i>Mystromys albicaudatus</i>	South Africa	S. Afric. Publ. H. Rep. f Year end. June 30 1927, 37.

	Name:	Locality:	Reference:
MURIDAE	13. <i>Pelomys fallax</i> <i>iredescens</i>	East Africa	Lurz, Arch. f. Schiffs- & Tropen-Hyg., 1913, 17, 593.
	14. Tree rat <i>Epimys</i> <i>dolichurus</i>	East Africa	Lurz, l.c.
	15. <i>Arvicanthis</i> <i>abyssinicus</i> <i>nubilans</i>	East Africa	Buchanan, Bull. Off. Internat. d'Hyg. Publ., 1925, 17, 492.
	16. Hamster rat <i>Cricetomys</i> <i>gambianus</i>	Gold Coast	Graham in Simpson, Rep. on Pl. in the Gold Coast in 1908, 21.
		Senegal	Laveau, Bull. Soc. Pathol. Exot., 1919, 12, 482 & Leger, Ann. de Med. & Pharm. Colon., 1926, 24, 273.
	17. African bush rat <i>Gelunda</i> <i>campanae</i>	Senegal	Leger & Baury, l.c.
	18. Field rat <i>Arvicanthis</i> <i>niloticus</i>	Egypt	Egypt. Plague Rep., Cairo 1923, 52.
	19. <i>Bandicota</i> <i>malabarica</i>	Ceylon	Philip & Hirst, Jl. Hyg., 1915, 15, 543.
	20. Bandicoot <i>Bandicota indica</i> (<i>Nesocia</i> <i>bandicota</i>)	India	Jl. Hyg., 1907, Pl. No., 760 & 1910, Pl. No., 459.
	21. Mole rat <i>Gunomys</i> <i>bengalensis</i>	India	Hossack, Jl. & Proc. Asiat. Soc. of Bengal, New Series 1906, 5.
Gerbillinae	22. <i>Gunomys</i> <i>gracilis</i>	Ceylon	Hirst & Vadivelu, The Rat-flea Survey of Kandy, Colombo, 1929 & person. inform. from Dr. Hirst.
	23. Sand mouse <i>Rhombomys</i> <i>opimus</i>	Turkestan	Nikanoroff, quot. by Grek-off, Guig. & Epid., 1924, 3, No. 4,50; Vj. Microb. & Epid., 1924, 3, 201; person. inform. from Pr. Nikanoroff.
		Transcaspia	Nikanoroff, Vj. Microb. & Epid., 1927, 6, 3.
	24. <i>Gerbillus</i> <i>tamaricinus</i>	South-East Russia	Golov & Ioff, Transact. of 1927 All-Russ. Anti-Pl. Conf., 110, 141.

	Name:	Locality:	Reference:
Gerbillinae	25. <i>Gerbillus meridianus</i>	South-East Russia	Golov & Ioff, l.c.
	26. Gerbille <i>Tatera lobengulae</i>	South Africa	Mitchell & Haydon, l.c. (1921).
	27. Namaqua gerbille <i>Desmodillus auricularis</i>	South Africa	Mitchell, S. Afric. Publ. H. Rep. f. Year end. June 30, 1926.
	28. Gerbille <i>Taterona schinzi</i>	South-West Africa	Fourie, l.c.
Microtinae	29. Field mouse <i>Microtus arvalis</i>	South-East Russia	Damberg & Tikhomiroff, quot. by Koltzov, Vrach. Gaz., 1915, 335; Nikanoroff, Vj. Microb. & Epid., 1922, 1, 71 & person. inform.
	30. Field mouse <i>Microtus socialis</i>	South-East Russia	Koltzov, l.c. (1917).
	31. Vole <i>Lagurus lagurus</i>	South-East Russia	Kniazevski & Grishina, Transact. of 1927 All-Russ. Anti-Pl. Conf., 87.
	32. <i>Ellobius talpinus</i>	South-East Russia	Gaiski, Vj. Microb. & Epid., 1931, 10, 59.
	33. <i>Microtus Brandti</i>	Transbaikalia	Skorodumoff, l.c. 82; Guig. & Epid., 1929, 8, No. 8.
MURIDAE	34. <i>Microtus Raddei</i>	Transbaikalia	Skorodumoff, ibid.
	35. Eastern Karroo rat <i>Paratomys luteolus</i>	South Africa	Mitchell l.c. (1924).
	36. Broom's Karroo rat <i>Myotomys broomi</i>	South Africa	Pirie, The Pl. Probl. in S. Africa, Publ. of the S. Afric. Inst. f. Med. Res., 1927.
	37. Cuvier's Karroo rat <i>Myotomys unisulcatus</i>	South Africa	1929 Rep., S. Afric. Inst. f. Med. Res.
Otomyinae			

	Name:	Locality:	Reference:
MURIDAE	Crice- tinae		
	38. Hamster <i>Cricetus cricetus</i>	South-East Russia	Koltzov, Zabolotny's Rep. on Pl. in S.-E. Russia, Leningrad, 1926.
	Dendro- myinae		
	39. Large-eared mouse <i>Malacothrix typicus</i>	South Africa	Mitchell, l.c. (1924).
Neoto- minae	40. Dusky-footed wood rat <i>Neotoma fuscipes</i>	California	McCoy & Smith, Jl. Inf. Dis., 1910, 7, 368.
	Sigmon- todinae		
JACULIDAE	41. Field rat <i>Hesperomys pulustris</i>	New Orleans	William, Americ. Jl. Publ. H., 1920, November.
	42. Jerboa <i>Alactaga mongolica</i>	Transbaikalia	Skorodumoff, Guig. & Epid., 1928, 7, No. 5, 69.
	43. Large jerboa <i>Alactaga saliens</i>	South-East Russia	Berdnikov, l.c.
	44. Small jerboa <i>Alactaga elater</i>	South-East Russia	Koltzov, l.c. (1926).
LEPORIDAE	45. Hare <i>Lepus timidus</i>	Transcaspia	Ignatiev, Vj. Microb. & Epid., 1927, 6, 160.
	46. Karroo hare <i>Lepus saxatilis</i>	South Africa	Pirie, l.c.
	47. Zulu hare <i>Lepus zuluensis</i>	South Africa	Pirie, l.c.
	48. Hare <i>Lepus europaeus</i>	England	Bulstrode, Loc. Govt. Bd. Rep., 1911, New Series, No. 52, 36; Martin & Rowland, Observat. on Rat Pl. in East Suffolk, ibid.
	49. Rabbit <i>Oryctolagus cuniculus</i>	England	Martin & Rowland, l.c.

CAVIIDAE

Name:	Locality:	Reference:
50. Guinea pig <i>Cavia cobaya</i>	Sydney	Thompson, Rep. of the Bd. of Health on a 2nd Outbr. of Pl. at Sydney, 1902.
	India	Liston, Jl. Bombay Nat. Hist. Soc., 1905, 16, 253 & Jl. Hyg., 1908, 7, 891.
	Manila	Schoebl, Phil. Jl. Sc., 1913, 8, 417.
	Senegal	Noc, Rep. sur le Fonct. du Lab. de l'A.O.F. en 1919, Dakar, 1920.
51. "Cuis" <i>Cavia aperea</i>	Argentine (a)	Uriarte & Gonzalez, C. R. Soc. Biol., 1924, 91, 1040.
	Ecuador	Eskey, U.S. Publ. H. Rep., 1930, 45, 2077.
52. Porcupine <i>Hydrochoerus capybara</i>	Mysore (India)	Bruce Low, Loc. Govt. Bd. Rep., 1898-01, 317.

UNCLASSIFIED
PEDETTIDAE

53. Spring hare <i>Pedetes caffer</i>	South Africa	Mitchell, l.c. (1924 Rep.)
54. Field rat <i>Mus rufinus</i>	Senegal	Lefrou, Bull. Off. Internat. d'Hyg. Publ., 1930, 22, 2106.

-(a) A *Ctenomys* is also suspected.

II. LIST OF WILD RODENTS PRESUMABLY SUFFERING FROM SPONTANEOUS PLAGUE

(NB. These could not be embodied in the main table because
A. The evidence as to infection is incomplete, and/or
B. Their species is not definitely determined).

	Name:	Locality:	Reference:
SCIURIDAE	1. Tarabagan ? <i>Arctomys robustus</i>	Kansu and Tibet	Skchivan, Arch. Russ. de Pathol., 1901, 6, 603; Parry China Med. Jl., 1918, 32, 86. (A, B)
	2. Marmot ? <i>Arctomys centralis</i>	Narynsk District	Tikhomirov, quot. by Grekoff, Med. Mysl Usbekistana, 1928-29, No. 3, 40 & Kalina, Ztrlbl. f. Bakt., Orig., 1929, 114, 50. (B)
	3. Sisel <i>Citellus mongolicus umbratus</i>	Tungliao District	Ando, Kurauchi & Nishimura, Kitasato Arch. of Exp. Med., 1931, 8, 24 & Bull. Off. Internat. d'Hyg. Publ., 1931, 23, 1952. (A)
	4. Palm rat	Senegal	Laveau, Bull. Soc. Path. Exot., 1919, 12, 291. (B)
MURIDAE	Murinae	5. Mole rat <i>Gunomys kok</i>	India Pandit, George, Mankikar & Natarajan, Ind. Jl. Med. Res., 1930, 17, 1223. (A)
		6. <i>Acomys cahirinus</i>	Egypt Wakil, The Third Pandemic of Pl. in Egypt, Cairo, 1932 (A)
	Gerbillinae	7. Gerbille*	Tunis Gobert, Ann. d'Hyg. Industr. & Soc., 1931, 9, 614. (A, B)
		8. Vole <i>Arvicola abyssinicus</i>	Kenya Symes & Hopkins, Col. & Prot. of Kenya Med. Dept. Rec. of Med. Res. Lab. No. 1, 1932. (A)
	Microtinae	9. <i>Microtus</i> §	Angola Froilano de Mello, Transact. 7th Congr. Far East. Assoc. Trop. Med., 1927, 2, 83. (A, B)

JACULIDAE	Name:	Locality:	Reference:
	10. Jerboa <i>Dipodipus sagitta</i>	South-East Russia	Nikanoroff, Bull. Off. Internat. d'Hyg. Publ., 1928, 20, 537. (A B)
	11. Jerboa	Baku District	Milman, Russki Vrach, 1915, 351. (B)
LEPORIDAE	12. Hare	Senegal	Lefrou, Bull. Soc. Path. Exot., 1929, 22, 517. (A, B)
		South Kirghisia	Kalina, Vj. Microb. & Epid., 1930, 9, 549 & 1931 10, 69. (A, B)
		Alma Atinsk District	Syssine, Bull. Off. Internat. d'Hyg. Pub., 1930, 22, 2101. (A, B)
UNCLASSIFIED	13. Field rat	Tunis	Gobert. Arch. Inst. Pasteur de l'Afrique du Nord, 1921, 1, 440. (A, B)
	14. Field rat	Rhodesia	Kinghorn, 1918, quot. Trop. Dis. Bull., 13, 324. (B)
	15. Field rodent	Azores	L.O.N. Wkly. Epid. Rec., 1931, 6, 726 & U.S. Publ. H. Rep., 1932, 47, 460. (A, B)
	16. Field rodent (mouse)	Khorassan (Persia)	Grekoﬀ, quot. by Clemow, Lancet 1913, 1, 1697 & Guig. & Epid., 1924, 3, No. 4, 50. (B)
	17. <i>Mus tamaricinus lasurus</i>	South-East Russia	Nikanoroff, l.c. (A. B)

Remarks.—(*) Gerbilles apparently also play a role in other parts of North Africa (Jorge, Bull. Off. Internat. d'Hyg. Publ., 1927, 19, 1257).
(§) *M. coucha* seems also involved.
(||) Plague infection was also proved in some mice (species?)

III. LIST OF RODENTS IN WHICH NO NATURAL PLAGUE HAS BEEN
FOUND BUT WHICH ARE SUSCEPTIBLE TO ARTIFICIAL INFECTION

Revised to December, 1932

<i>Name:</i>	<i>Locality:</i>	<i>Reference:</i>
1. <i>Mus agrarius</i>	Formosa	Kuraoka, Transact, 3rd Congr., Far East. Assoc. Trop. Med., 1913, 204.
2. Field Mouse (a)	Formosa	Kuraoka, ibid.
3. Striped Hamster <i>Cricetulus griseus</i>	China (Chihli)	Hsieh, National Med. Jl., 1919, 5. 20.
4. Sisel <i>Citellus mongolicus</i> (b)	South Manchuria	Wu Lien Teh & Eberson. Jl. Hyg., 1917, 16, 1.
5. Hamster <i>Cricetulus furunculus</i>	Transbaikalia	Jettmar, Jl. of Transbaik. Med. Soc. 1922, No. 2, 95 & Sukneff, Publ. of Harbin Med. School, No. 1, 1922, 213.
6. Rat-Hare <i>Ochotona dauricus</i>	Transbaikalia	Jettmar & Sukneff, ibid.
7. <i>Spermophilus eversmanni</i>	Transbaikalia	Jettmar, Ztschr. f. Hyg. & Infekt.-Kr. 1923, 97, 329.
8. <i>Citellus guttatus</i>	South-East Russia	Shurupoff, Russki Vrach, 1911, 1301 and Zentralbl. f. Bakt., 1912, 65, 243.
9. Marmot <i>Marmota bobac</i> (c)	South-East Russia	Golov & Joff, Transact. 1927 All-Russ. Anti-Pl. Conf., 102.
10. Jerboa <i>Dipodipus sagitta</i> (d)	South-East Russia	Golov & Joff, ibid.
11. <i>Micromys minutus</i>	South-East Russia	Golov & Joff, ibid.

<i>Name:</i>	<i>Locality:</i>	<i>Reference:</i>
12. <i>Arvivola</i> <i>amphibius</i>	South-East Russia	Golov & Joff, <i>ibid.</i>
13. <i>Mesocricetus</i> <i>eversmanni</i>	South-East Russia	Golov & Joff, <i>ibid.</i>
14. <i>Dyromys</i> <i>nitedula</i>	South-East Russia	Golov & Joff, <i>ibid.</i>
15. Alpine marmot <i>Marmota</i> <i>marmota</i>	Europe	Wurtz, quot. by Dujardin- Beaumetz & Mosny; Dujar- din-Beaumetz & Mosny, C. R. Acad. Sci. 1912, 155, 329.
16. Lerot (Door mouse) <i>Myoxys murinus</i>	Senegal	Leger & Baury, C. R. Acad. Sci., 1922, 175, 734.
17. <i>Acomys cahirinus</i> (d)	Egypt	Todd in Petrie, Progress Rep. on the Work of Pl. Invest. Staff in Upper E., 1911-12, Cairo, 1912, 20.
18. Jerboa	Egypt	Todd, <i>ibid.</i>
19. "Root rat" <i>Tachyoryctes</i> <i>daemon.</i>	East Africa	Lurz, Arch. f. Schiffs & Tropenhyg., Sept. 1913, 17, 593.
20. Dwarf mouse <i>Leggada</i>	South Africa	Pirie, Publ. of the S. African Inst. f. Med. Res., 1927, No. 20, 122.
21. Fat mouse <i>Steatomys krebsi</i>	South Africa	Pirie, <i>ibid.</i>
22. Grey mole-rat <i>Cryptomys sp.</i>	South Africa	Pirie, <i>ibid.</i>
23. Water rat <i>Otomys irroratus</i>	South Africa	Pirie, <i>ibid.</i>
24. Cape hare <i>Lepus capensis</i>	South Africa	Pirie, <i>ibid.</i>
25. Field mouse <i>Microtus</i> <i>californicus</i>	California	McCoy, Jl. Inf. Dis., 1909, 6, 283.

Name:	Locality:	Reference:
26. California pocket gopher <i>Thomomys bottae</i> (e)	California	Ibidem & Jl. Inf. Dis., 1911 8, 42.
27. Chipmunk <i>Callospermophilus</i> (<i>Citellus</i>) <i>chrysodeirus</i>	California	McCoy, ibid, 1911.
28. <i>Ammospermophilus</i> <i>leucurus</i>	California	McCoy & Chapin, U. S. Publ. H. Bull. No. 53, 1912, 15.
29. Rock squirrel <i>Citellus grammurus</i>	New Mexico	McCoy & Smith, Jl. Inf. Dis., 1910, 7, 374.
30. Arizona prairie dog <i>Cynomys ludovician.</i> <i>arizonensis</i>	New Mexico	McCoy & Smith, ibid.
31. Eastern desert wood rat <i>Neotoma albifula</i> <i>angusticeps</i>	New Mexico	McCoy & Smith, ibid.

- Remarks.—(a) "Field and forest mice" were found susceptible by Nuttall (Ztrbl. f. Bakt., 1897, 22, 87).
 (b) Preliminary experiments were made by Strong (Rep. of Internat. Pl. Conf., Mukden, 1911, 239) and by Shibayama (ibid., 31). Nishimura (Jl. Orient. Med., 1930, 13, 7) experimented upon the *Citellus mongolicus ramosus* Thos.
 (c) Experiments with the Ural tarabagan were carried out in 1903 by Shurupoff (Russki Vrach, 1911. No. 33, 1301). Flu (Genéesk. Tijdschr. v. Ned.-Indie, 1914, 54, 540) performed experiments with marmots (?).
 (d) Possibly found natural infected.
 (e) "Gophers are not sufficiently susceptible to infection with *B. pestis* to be of any importance from an epidemiological point of view" (McCoy).

IV. FLEAS OF WILD RODENTS SUFFERING FROM
SPONTANEOUS PLAGUE.

<i>Locality:</i>	<i>Host:</i>	<i>Fleas:</i>
Transbaikalia and Mongolia	<i>Arctomys bobac</i>	<i>Oropsylla silantiewi</i> *
	<i>Alactaga mongolica</i>	<i>Ceratophyllus</i> sp.
	<i>Spermophilus dauricus</i>	<i>Ceratophyllus tesquorum</i> *
	<i>Microtus Brandti</i> & <i>Raddei</i>	<i>Ceratophyllus</i> sp.
India	<i>Gunomys bengalensis</i>	<i>Xenopsylla astia</i> * <i>X. cheopis</i> *
	<i>Sciurus palmarum</i>	<i>Xenopsylla cheopis</i> * <i>Ceratophyllus argutus</i>
Transcaspia	<i>Rhombomys opimus</i>	<i>Xenopsylla gerbilli</i> <i>X. hirtipes</i> <i>X. skrjabini</i> <i>Coptopsylla</i> sp. <i>Ceratophyllus fidus</i> <i>C. laeviceps</i> * <i>C. tersus</i> <i>Ophthalmopsylla volgensis</i> * <i>Ctenophthalmus dolichus</i> <i>Rhadinopsylla cedestis</i>
South-East Russia	<i>Citellus pygmaeus</i>	See Table V.
	<i>Citellus fulvus</i>	<i>Oropsylla ilovaiskii</i> *
	<i>Jaculidae</i> (<i>Dipodidae</i>)	<i>Ophthalmopsylla volgensis</i> * <i>O. kasakiensis</i> * <i>Mesopsylla hebes</i> * <i>M. lenis</i> * <i>M. tuschkan</i>
	<i>Microtinae</i>	<i>Ceratophyllus consimilis</i> * <i>Amphipsylla rossica</i> <i>A. prima</i>
	<i>Gerbillinae</i>	<i>Xenopsylla mycerini</i> * <i>Ceratophyllus laeviceps</i> *
	<i>Cricetus cricetus</i>	<i>Ctenophthalmus breviatus</i> <i>Ct. orientalis</i> <i>Ct. wagneri</i>

<i>Locality:</i>	<i>Host:</i>	<i>Fleas:</i>
	<i>Ellobius talpinus</i>	<i>Xenopsylla mycerini*</i>
England	<i>Oryctolagus cuniculus</i>	<i>Spinopsyllus cuniculi*</i> <i>Ceratophyllus fasciatus*</i>
	<i>Lepus europaeus</i>	<i>Spinopsyllus cuniculi*</i>
South Africa	<i>Tatera lobengulae</i>	<i>Dinopsyllus lypusus*</i> <i>Chiastopsylla rossi*</i> <i>Xenopsylla eridos*</i> <i>X. hirsuta</i> <i>X. trifarius</i> <i>X. lobengulae</i> <i>Listropsylla stygius</i> <i>Ctenophthalmus calceatus</i> <i>Leptopsylla musculi*</i>
	<i>Rattus coucha</i>	<i>Dinopsyllus lypusus*</i> <i>Xenopsylla eridos*</i> <i>Chiastopsylla rossi*</i> <i>Xenopsylla brasiliensis*</i> <i>Pulex irritans*</i>
	<i>Rhabdomys pumilio</i>	<i>Dinopsyllus lypusus*</i> <i>Xenopsylla eridos*</i> <i>X. versuta</i> <i>Chiastopsylla rossi*</i> <i>Listropsylla agrippinae</i> <i>L. chelurae</i> <i>Chiastopsylla octavii</i> <i>Dinopsyllus longifrons</i>
	<i>Malacothrix typicus</i>	<i>Dinopsyllus lypusus*</i> <i>Xenopsylla eridos*</i> <i>Listropsylla stygius</i>
	<i>Geosciurus capensis</i>	<i>Xenopsylla erilli*</i> <i>Ctenocephalus canis*</i> <i>Echidnophaga bradyta</i> <i>E. gallinacea</i> <i>Xenopsylla eridos*</i> <i>Dinopsyllus lypusus*</i> <i>Pulex irritans*</i>
	<i>Paratomys luteolus</i>	<i>Chiastopsylla pitchfordi</i> <i>Listropsylla agrippinae</i> <i>Dinopsyllus longifrons</i> <i>Xenopsylla eridos*</i> <i>Chiastopsylla rossi*</i> <i>Ch. quadrisetus</i>
	<i>Pedetes caffer</i>	<i>Xenopsylla caffer</i>
	<i>Desmodillus auricularis</i>	<i>X. eridos*</i> <i>Chiastopsylla rossi*</i> <i>?Dinopsyllus lypusus*</i>

Locality:	Host:	Fleas:
	<i>Myotomys broomi</i>	<i>Chiastopsylla mulleri</i> <i>Listropsylla agrippinae</i> <i>Chiastopsylla rossi*</i> <i>Xenopsylla eridos*</i> <i>Hypsophthalmus aganippes</i>
	<i>Lepus zuluensis</i> & <i>saxatilis</i>	<i>Ctenocephalus canis*</i> <i>Xenopsylla eridos*</i>
	<i>Mystromys albicaudatus</i>	<i>Dinopsyllus lypusus*</i> <i>Chiastopsylla rossi*</i> <i>Xenopsylla cheopis*</i>
South-West Africa	<i>Taterona schinzi</i>	<i>Xenopsylla eridos*</i>
Central Africa	<i>Rattus coucha</i> <i>ugandae</i>	<i>Xenopsylla brasiliensis*</i> <i>X. cheopis*</i> <i>Dinopsyllus lypusus*</i>
	<i>Arvicanthis abyssinicus</i> <i>nubilans</i>	<i>Xenopsylla brasiliensis*</i> <i>Dinopsyllus lypusus*</i> <i>Xenopsylla cheopis*</i>
	<i>Pelomys fallax iredescens</i> & <i>Epimys dolichurus</i>	<i>Xenopsylla cheopis*</i> <i>Leptopsylla musculi*</i> <i>Ceratophyllus fasciatus*</i>
Belgian Congo	<i>Mastomys ugandae</i>	<i>Xenopsylla brasiliensis*</i> <i>X. cheopis*</i> <i>Dinopsyllus lypusus*</i> <i>Ctenophthalmus n. sp.</i> <i>?Ct. cabirus</i> <i>Leptopsylla segnis*</i>
Gold Coast	<i>Cricetomys gambianus</i>	<i>Xenopsylla aequisetosus</i> <i>X. nubicus</i> <i>X. cheopis*</i>
French West Africa	<i>Mastomys coucha</i> <i>Cricetomys gambianus</i> <i>Golunda campanae</i> <i>Mus rufinus</i>	Mainly <i>X. cheopis*</i>
Upper Egypt	<i>Arvicanthis niloticus</i>	<i>Xenopsylla cheopis*</i>
California	<i>Citellus beechyi</i>	<i>Ceratophyllus acutus*</i> <i>Hoplopsylla anomalus*</i>
Ecuador	Guinea pig	<i>Rhopalopsyllus cavicola</i> <i>Hectopsylla Suarez</i> <i>X. cheopis*</i>

Remark.—*Known to bite man.

V. PARASITES OF THE CITELLUS PYGMAEUS

(As compiled by Sassuchin & Tiflov, Vj. Microb. & Epid., 1932, 11,130)

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- A. Intestinal Parasites
- Protozoa:*
1. *Entamoeba citelli* Becker, 1926*
 2. *Chilomastix magna* Becker, 1926*
 3. *Trichomonas muris*, v. *citelli* Becker, 1926
 4. *Tetratrichomastix citelli* Becker, 1926*
 5. *Octomitus pulcher* Becker, 1926*
 6. *Giardia beckeri* Hegner, 1926
 7. *Eimeria citelli* Kartchner & Becker, 1930*
 8. *Eimeria volgensis* Sassuchin & Rauschenbach, 1932*
- Vermes:*
9. *Plagiorchis maculosus citelli* Schulz, 1931
 10. *Hymenolepis ognevi* Skrj., 1924
 11. *Ascaris ioffi* Schulz, 1931
 12. *Gongylonema longispiculum* Schulz, 1927
 13. *Arduenna kutassi* Schulz, 1927
 14. *Protospirura suslika* Schulz, 1927
 15. *Trichostrongylus instabilis* Raill., 1893
 16. *Trichostrongylus extenuatus* Raill., 1898
 17. *Trichostrongylus probolurus* (Raill., 1896) Looss, 1905
 18. *Haemonchus contortus* (Rud. 1803) Gobb, 1898
 19. *Nematodirus mugosaricus* Schulz, 1926
 20. *Eucoleus baskakovi* Schulz, 1928
 21. *Moniliformis moniliformis* Bremser, 1811
 22. *Trichocephalus* sp.
- B. Blood Parasites
- Protozoa:*
23. *Trypanosoma spermophili* Laveran, 1911*
 24. *Toxoplasma nikanorovi* Sassuchin & Gaiski, 1930
 25. *Leucocytoegregarina merechkowsky* Tartakovski, 1914
 26. *Piroplasma kolzovi* Sassuchin, 1930*
 27. *Grahamia hegneri* Sassuchin, 1930
- C. Ectoparasites
- Arthropoda*
- Acarinae:*
28. *Ixodes redikorzevi*, v. *lagurae* Olen., 1929*
 29. *Rhipicephalus schulzei* Olen., 1929*
 30. *Dermacentor niveus* Neum., 1897
 31. *Hyalomma* sp.
 32. *Haemaphysalis numidiana* Neum., 1905
- Aphaniptera:*
33. *H. cinnabarina punctata* Can. & Fanz., 1877
 34. *Ceratophyllus tesquorum* W., 1898*
 35. *Frontopsylla semura* W. & I., 1926*
 36. *Neopsylla setosa* W., 1898*
 37. *Ctenophthalmus breviatus* W. & I., 1926*
 38. *Ctenophthalmus pollex* W. & I., 1926*
 39. *Oropsylla ilovaiskii* W. & I., 1926
 40. *Pulex irritans* L., 1758
 41. *Ctenocephalus canis* C., 1826
 42. *Ctenocephalus felis* B., 1835
 43. *Ceratophyllus mokrzeckyi* W., 1915
 44. *Ophthalmopsylla volgensis* W. & I., 1926
 45. *Amphipsylla rossica* W., 1912
 46. *Mesopsylla lenis* J. & R., 1915
 47. *Mesopsylla hebes* J. & R., 1915
 48. *Mesopsylla tuschkan* W. & I., 1926
 49. *Ctenophthalmus orientalis* W., 1898
 50. *Ctenophthalmus secundus* W., 1916.
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Remark.—*Means commonly found.

EARLY CHINESE TRAVELLERS AND THEIR SUCCESSORS*

BY WU LIEN-TEH

I deeply appreciate the honour which the Council of the Royal Asiatic Society has conferred upon me by asking me to deliver the first lecture in these newly-completed premises of the Society.

In choosing my subject "Early Chinese Travellers" I am mindful of the fact that it covers a wide field, that authorities to be consulted, whether Chinese or foreign, are few and scattered, and that the data, even when collected, are not quite well-balanced, for some parts are too short, others too long and the rest too uncertain. However, with your permission I will try during the next half-an-hour at my disposal to entertain you with the results of my humble search for Chinese travellers of the past, who have ventured outside their flowery land in search of faith, gold, learning, power, conquest, love and other human desires.

I have here a list of thirty-five persons of both sexes commencing from B.C. 500 (Eastern Chou dynasty) until about fifty years ago before the break-up of the Ch'ing dynasty.

1. MENG CHIANG (孟姜).

So far as I can ascertain, the first Chinese to have travelled outside the confines of the China of those days was a lady—Meng Chiang (孟姜) by name—who was married to an official named Ch'i Liang (杞梁) serving under the Eastern Chou dynasty (B.C. 500). Unfortunately, three days after their wedding, the bridegroom disappeared, and she was told that for political reasons he had been exiled for an indeterminate period to Ch'ang Ch'eng (長城). Meng Chiang waited in vain for his return, and after years of uncertainty resolved to find him. One winter she prepared thick clothing and set out with an old male servant on a long journey. It was said that Meng Chiang travelled thousands of li—over mountains, across rivers and desert plains—as far as the borders of present Mongolia before she reached her destination, only to discover that her husband had died years previously and been buried under the city wall. She wept day and night on the spot, and on the

*Inaugural address delivered on the occasion of the opening of the new building of the Royal Asiatic Society (North China Branch). February 23rd, 1933.

seventh day part of the mud wall collapsed revealing the apparent remains of her husband. The sorrowful widow collected the bones and brought them back to his home, after which she drowned herself in the Tse River. 溜水

2. HSU SHIH 徐市 (B.C. 221-210).

The second traveller was Hsu Shih or Hsu Fu, a Taoist monk who was commissioned by Ch'in Shih Huang-ti 秦始皇帝, builder of the Great Wall and the emperor who first united the loose feudal states of China into a conglomerate whole, to proceed to the three fairy isles in the East Sea and search for the elixir of life. These islands were known as P'englai 蓬萊, Fangchang 方丈 and Yinchou 瀛洲. Hsu Shih fitted out an expedition consisting of several large sailing vessels, carrying on board beside his retinue about three thousand youths and maidens as offerings to the fairies. But the party never returned, nor was it heard of afterwards. It is believed by Chinese that these youthful travellers to the Eastern Seas were among some of the earliest ancestors of the Japanese, whose descendants unto this day possess names corresponding to Chinese surnames, such as, Lin 林 (Hayashi); Ch'in 秦 (Hata); Yuan 原 (Hara); Wu 吳 (Kure); T'ien 田 (Ta); Tung 東 (Higashi); Nan 南 (Minami); Hsi 西 (Nishii), etc.

3. CHANG CH'IENT 張騫 (B.C. 138-127).

Between the third and second centuries B.C. three nomadic tribes of Mongolian origin, namely, Yueh-chi, Hiung-nu and Wu-sun were constantly warring for power and territory over a wide area of land stretching from present West Kansu over Hindu-kush. Bactria, Sogiana, Afghanistan, Herat, Kandahar, Sind, Ferghana to the Oxus Valley near modern Bokhara. Emperor Wu Ti of the Han dynasty, who himself had had incessant troubles with the Hiung-nu (Huns of European history), sent in B.C. 128 his minister Chang Ch'ien accompanied by a large retinue as ambassador with presents of silk to many of the powers of West Central Asia. On the way—only the land route was then known—he was taken prisoner by the Hiung-nu, and spent almost eight years of rather pleasant captivity among them. During this time he was able to study carefully the geography and habits of the inhabitants and send important information to his emperor for future use. Finally Chang Ch'ien escaped to Ta-yuan (Ferghana), whose people "having heard of the wealth and fertility of China had tried in vain to communicate with it." Here Chang Ch'ien saw certain bamboo and cloth goods of Chinese-make and was told they had been brought in through India (Yen-tu). It appeared that Chinese wares had reached India by indirect trading through the primitive tribes from the *south-west* of China,

whereas Chang Ch'ien had himself taken the *north-west* route. Plans were accordingly laid before Wu Ti.

So it was resolved to open up a north-western route, and war was resumed on a grand scale against the Hiung-nu. Operations were almost immediately successful, the Western Horde of the Hiung-nu was severely defeated and driven away to the north, and Chinese dominion was advanced as far as Lop-nor (B.C. 121). The way being now clear, embassies were despatched during the next few years to all the countries mentioned by Chang Ch'ien in his report, and the diplomatic ambitions of Wu Ti were to a great extent realized. The *Shih-chi* or official records relate: 'Such missions would be attended by several hundred men, or by a hundred men, according to their importance. At least five or six missions were sent out in the course of a year, and as a rule more than ten; those sent to distant countries would return home after eight or nine years, those to nearer ones within a few years.' Chang Ch'ien had reported that there was no sericulture in the western lands, so that silk formed a large part of the ambassadorial gifts; those to the Wu-sun were specified as silk and gold.

The climax of Wu Ti's forward policy in Central Asia was the conquest of Ferghana by the war of B.C. 104-100. The Emperor sent presents to the king of Ferghana and requested in return a number of the special breed of horses for which his realm was famous. But Ferghana was by this time 'overstocked with Chinese produce,' and there was reluctance to give away the precious horses. Deeming that Chinese power could not reach them, the king and magnates of Ferghana rejected the demand; the Chinese envoys used insulting language, and were in retaliation murdered at the frontier on their return journey. A Chinese army under a general named Li Kuang-li 李廣利 was sent to avenge them, but it was driven back after many of the troops had perished from starvation in the Tarim deserts. Wu Ti, however, refused to admit defeat. 'The Emperor thought that having dispatched an unsuccessful expedition against Ferghana, a small country, would cause Bactria and other neighbouring states to feel contempt for China, and that the pedigree horses of Ferghana would never be forthcoming.' A new army was therefore sent out—'60,000 men not counting those who followed as carriers of extra provisions; 100,000 oxen; more than 30,000 horses, myriads of donkeys, mules and camels, and a commissariat well-stocked with supplies besides cross-bows and other arms. All parts of the empire had to bestir themselves in making contributions.' This force overcame the desert by dividing into columns which took different routes; on reaching Ferghana it won a decisive

victory and obtained the submission of the country. Subsequently 'China sent more than ten embassies to countries west of Ferghana to collect curiosities and at the same time to impress upon such countries the importance of the victory over Ferghana.'

Thus by the end of the second century B.C., within twenty-eight years of the discovery by Chang Ch'ien of China's 'New World,' Chinese arms had penetrated triumphantly west of the Pamir divide, and regular intercourse with Western Asia had been established. And now through Western Asia an indirect trade gradually developed, linking China with Europe. During the early years of the first century the use of silk, which at the Parthian court probably dated from the coming of the first Chinese embassy, spread from Parthia to the Mediterranean. The taste won its way to Europe at a time when the unification by Rome of the whole Mediterranean world had given unprecedented stimulus to industry and commerce and had created an enormously rich ruling class with an appetite for every kind of exotic luxury.

In Ferghana, in the valley of the upper Iaxartes, the army of Li Kuang-li advancing from the east encamped perhaps on the very ground which 227 years before had seen the tents of Alexander of Macedon. The great march of Alexander from the Hellespont to the Pamirs and the Punjab spread Greek settlements over Western Asia and brought Mesopotamia, Iran and India into the one world of intercommunication with the lands of the Mediterranean. But it did nothing to open a road to China, which remained separate, secluded, utterly unknown, as it had been on another planet. Between Alexandria the Furthest and the Chinese pale in Kansu there intervened such a barrier of natural wilderness as did not anywhere confront the traveller between Spain and Bengal. A modern traveller has called the Takla-makan 'the most appalling desert on the face of the earth.' The bridging of this gap was accomplished not from the west but from the east, not by the Persians or by the Greeks but by the Chinese themselves. It was the Chinese who, first by an exploring diplomacy and then by force of arms, broke through to the land which alike for Achaemenid and Macedonian had been nothing but a cul-de-sac. Chang Ch'ien had indeed done well!

4. WANG CHAO CHUN 王昭君 (B.C. 40).

We may now turn to Wang Chao Chun, one of the four historical beauties of China. Chao Chun (the name by which she is usually known) was one of the hundreds of maidens sent to the imperial court of Yuan Ti 元帝 (B.C. 48-33) to be selected as imperial concubines, but though most beautiful

was unfortunate enough to incur the displeasure of the Court painter Mao Yen-shao 毛延壽 who, because the father had not paid him sufficient *largesse*, purposely added a few blemishes to the portrait of the young applicant, thus making her homely instead of lovely. For three years she lingered in solitude in one of the deserted palaces and was only brought out when a threatening leader of the Hsiung-nu tribe applied for a Chinese bride to take to Mongolia. Yuan Ti then selected who he thought was the ugliest among the women-in-waiting in his palace for this occasion, and it was only when the incomparable Chao Chun knelt before the imperial presence to say good-bye, that Yuan Ti found how badly he had been deceived by his minister. But it was then too late and against his will he had to see the forlorn beauty ride off on her long trip to Mongolia. However, the unhappy exile did her duty and bore a son to the Mongolian Prince and brought him up until he was ready for the throne. Unfortunately at the death of the ruler Mongolian customs demanded that the new Prince should wed his own mother. This Chao Chun as a strict follower of Confucius could never follow; so she committed suicide. To this day Chao Chun has been regarded as the most virtuous of the four historical beauties and her ever-green graveyard may still be seen marked by a stone tablet in the city of Sui-yuan 綏遠.

5. PAN CH'AO 班超 (A.D. 32-102).
KAN YIN 甘英 (87).

We may skip the next two names, Pan Ch'ao and Kan Ying of the early Han dynasty. These were two famous generals sent by Ming Ti to western countries for purposes of pacification which work lasted 16 years. The latter reached Babylonia in A.D. 97 and made a treaty with its ruler. From these we now pass on to the early group of learned and adventurous Buddhists who took long trips overland to India in search of Buddhistic scriptures for use in China. Earliest among these was:

6. CHU SHIH-HSING 朱士行 A.D. 260

He lived during the troublous times of the Three Kingdoms. Chu could with fair accuracy be called the first Chinese priest to have proceeded far abroad. Starting from Loyang, the then capital, he reached Yu-t'ien (Khotan) in 260 after one year's difficult travelling. Here he found the new faith in a flourishing condition and obtained a sutra of ninety sections which Chufahu (a priest of the Getae nation) and other disciples translated into Chinese. He made his home at Khotan and died there. After him, there followed more than one hundred monks bent upon the same mission of study, namely, fifty-three

during the years 260-581 and fifty-two during the years 628-789.

7. FA HSIEN 法顯 (A.D. 399-414).

The next, Fa Hsien, whose name has been immortalised in the western world by the translations of Remusat, Beal, Legge and Giles, was a native of Ping-yang 平陽. He practically walked from Central China in the fourth century across the desert of Gobi over the Hindu-Kush, and then traversed India down to the mouth of the Hoogly, where he took ship. After fifteen years' absence he returned by sea to China, bringing back what he went forth to secure—books of the Buddhist canon and images of Buddhist deities. In his self-imposed mission, Fa Hsien was accompanied by similarly devout priests like Hui Ching, Tao Cheng, Hui Ying, Hui Wei and others. The party started from Chang-an, the capital, crossed the Lung division of Shensi and Kansu, reached the market town of Chang-yeh (Kanus), where more pilgrims joined them. At Tun-huang on the Great Wall, they obtained all necessities for braving the Gobi desert. Their impressions of this are thus:

'In this desert there are a great many evil spirits and also hot winds; those who encounter them perish to a man. There are neither birds above nor beasts below. Only the bleached bones of man and beast point the way.'

At Shan-shen (south of Lop Nor) they were received hospitably by the king who had adopted the Faith, and whose subjects practised the religion of India. Details of this historic journey are vividly described in Fa Hsien's *Record of Buddhistic Kingdoms*, such as, his visit to Hiro (where was situated a shrine containing Buddha's skullbone covered with precious stones); their trials when crossing the snowy mountains of Safed Koh, at which spot his companion Hui Ching developed frost-bite and asked to be left to die; the arrival of the two surviving pilgrims in Afghanistan (where lived 3000 priests); and later the land of the Brahmans (where the ruler used no corporal punishment but merely a fine according to the gravity of the offence, and where rooms, food and clothing were ever provided for resident and travelling priests). After staying at Pataliputra (Patna), where Asoka once ruled, they passed the Deccan and reached the Ganges, whose course they followed as far as the kingdom of Champa. From Tamluk (mouth of the Hoogly) Fa Hsien, now alone, embarked upon a merchant vessel for Kandy in Ceylon, where Buddha's tooth may up to this moment be seen. Fa Hsien found Ceylon full of Buddhistic relics and stayed there for two years. Finally, he took a vessel with 200 other souls on board bound for Java, but during

a storm the ship sprang a leak. Excitement reigned supreme for 13 days, during which progress was only made possible by reference to the sun, moon and constellations, the ship not keeping any definite course and being drifted at the mercy of the wind and wave. Thus they continued for 90 days and at last arrived in Java, 'where heresies and Brahamanism flourished.' After five months' stay, Fa Hsien took another vessel bound for Canton, but a heavy gale drove it to Ch'ang Kuang (modern Tsingtau) in Shantung, where they obtained fresh water and vegetables. Fa Hsien thus ended his wonderful narrative:

"I spent six years in travelling from Chang-an to Central India; there I stayed six years, and it took me three more to reach Ch'ing-chou. The countries I passed through numbered more than thirty. From the sandy desert (Gobi), westward all the way to India, the dignified deportment of the priesthood and the good influence of the Faith were beyond all praise. As the ecclesiastics at home had no chance to hear about these things, I gave no thought to my own unimportant life, but came home across the sea, encountering still more difficulties and dangers. Happily, I was accorded protection by the divine majesty of the precious Trinity, and was thus preserved in the hour of danger. Therefore I write down on these bamboo tablets and silk an account of what I have been through, desiring that the gentle reader should share my information."

8-12. SUNG YUN 宋雲, HUI SHENG 惠生, PEI CHU 裴矩
WEI CHIEH 韋節 and TU HSING MAN 杜行滿

Sung Yun and Hui Sheng were two pilgrims sent from Loyang by the Empress Hu 胡太后 in 518 to Hsi Yueh 西域 (Tibet). Pei Chu after travelling overland abroad wrote a book of Travels called Hsi Yueh Tu Chi 西域圖記. Wei Chieh and Tu Hsing-man served as envoys by Yang Ti of the Sui dynasty to the kingdom of Kashmir. Wei recorded his observations in a book known as Hsi Fan Chi 西蕃記 now lost.

13. HSUAN CHUANG 玄奘 (602-664).

In 629, that is, one year after the arrival of Muhamed's envoys at Canton, a learned and devout priest Hsuan Chuang started out from Chang-an, the capital of the Tangs under the great T'ai Tsung who was called by Gibbons the Augustus of the East. Because foreign travelling was then forbidden, Hsuan Chuang had to be cautious, but with the help of friends was able at last to leave his native land. He was away fourteen years and returned the same way that he left, in 645, when after declining official posts he devoted his remaining years

to the writing of his famous classic *Ta Tang Hsi Yueh-chi* 大唐西域記 or Memoirs of Western Lands.

Hsuan Chuang's journey was an enormous one. He went and came back by way of the Pamirs. He took the northern route, crossing the desert of Gobi, passing along the southern slopes of the T'ien Shan, skirting the great deep blue lake of Issik Kul, and so to Tashkend and Samarkand, and then more or less in the footsteps of Alexander the Great southward to the Khyber Pass and Peshawar. He returned by the southern route, crossing the Pamirs from Afghanistan to Kashgar, and so along the line of retreat the Yueh-chi had followed in the reverse direction seven centuries before, and by Yarkand, along the slopes of the Kuen Lun to rejoin his former route near the desert end of the Great Wall. Each route involved some hard mountaineering. His journeyings in India are now untraceable; he was there fourteen years, and he traversed the peninsula from Nepal to Ceylon. Among other things, he shows us the Turks in possession not only of what is now Turkestan, but all territory along the northern route. He mentions many cities and considerable cultivation. He is entertained by various rulers, allies of more or less nominal tributaries to China, and among others by the Khan of the Turks, a magnificent person in green satin, with his long hair tied with silk. The gold embroidery of this grand tent shone with a dazzling splendour; the ministers of the presence in attendance sat on mats in long rows on either side all dressed in magnificent brocade robes, while the rest of the retinue on duty stood behind. After a short interval the envoys from China and Kao-chang were admitted and presented their despatches and credentials, which the Khan perused. He was much elated, and caused the envoys to be seated; then he ordered wine and music for himself and then a grape-syrup for the pilgrim. Hereupon all pledged each other, and the filling and draining of the wine cups made a din and bustle, while the mingled music of various instruments rose loud: although the airs wore the popular strains of foreigners, yet they pleased the senses and exhilarated the mental faculties. After a little interval, piles of roasted beef and mutton were served for the others, and lawful food, such as cakes, milk, candy, honey, and grapes, for the pilgrims. After the entertainment, grapesyrup was again served and the Khan invited Hsuan Chuang to improve the occasion, whereupon the pilgrim expounded the doctrines of the Ten Virtues, compassion for animal life, and the paramitas and emancipation. The Khan, raising his hands, bowed, and gladly believed and accepted the teaching.

Hsuan Chuang's account of Samarkand is of a large and prosperous city, "a great commercial entrepôt, the country about

it very fertile, abounding in trees and flowers and yielding many fine horses. Its inhabitants were skilful craftsmen, smart and energetic." At that time we must remember there was hardly such a thing as a town in Anglo-Saxon England.

Hsuan Chuang brought back:

- (a) 115 grains or relics taken from Buddha's Chair;
- (b) one gold statue of Buddha, 3 ft. 3 in. in height, with a transparent pedestal;
- (c) another statue, 3 ft. 5 in. in height;
- (d) others of silver and carved in sandal-wood;
- (e) 124 sutras of the "Great Development." Altogether 657 total works were carried on 22 horses. Hsuan Chuang went to Ch'ang-an 長安 to translate, assisted by 12 monks. Nine others revised.

That Hsuan Chuang was a strong literalist as well as an accurate translator was proved by the fact that he did the 120 volumes entire, with all their wearisome reiteration of metaphysical paradoxes, as compared with the rather abbreviated translation of Kumarajiva, who omitted repetitions and superfluities.

Hsuan Chuang lived 19 years after his return; he completed 740 works in 1335 books.

14. WANG HSUAN-TS'E 王玄策 (646-661).

After the visit of Buddhist Hsuan Chuang 玄奘 to India, China was recognized as the world power by all the petty States of India. The king of Oudiyana 烏長國 the then leading state of North India, twice sent envoys together with valuable gifts. In 648, Emperor T'ai Tsung 太宗 appointed Wang Hsuan Ts'e 王玄策 as special envoy to Oudiyana with Chiang Shih-jen 蔣師仁 as assistant.

It happened that the king of Oudiyana had passed away and his kingdom was usurped by one of his followers. The usurper did not recognize Wang as an envoy from China and temporarily succeeded in expelling him, the latter's horsemen being all killed. With a view to punishing the usurper, Wang hurried to Thibet and Nepal to ask for military help. One thousand soldiers were sent from Thibet and seven thousand cavalry from Nepal. As a result the usurper was captured together with members of his family. This was the first and only war between India and China.

In 657, Hsuan T'se was sent again to India on a Buddhistic mission. He returned in 661. It is no exaggeration to say that Wang Hsuan-t'se was the most important figure in the old diplomatic relations between India and China.

15. I CHING 義淨 (671-695).

After Hsuan Chuang's death exploration of the west became the fashion during the T'angs, and another priest, I Ching by

name, undertook a trip *by the sea route* to India. I Ching was born at Fanyang (Chihli P.), started from P'an-yu 番禺 (Canton) in 671 in a merchant vessel and after a stormy voyage reached Bhoga at the mouth of the Ganges. He stayed over 20 years in India (671-695), studying every available aspect of Buddhism, and brought back some 400 texts, the slokas alone numbering 500,000, as well as a plan of the Diamond Seat of the sage.

16. WU-KUNG 悟空 (751).

Wu-Kung's was the last pilgrim to have travelled abroad in the T'ang dynasty. His original name was Chu Chao-feng 車朝奉. In 751 A.D. he was appointed an attache of the envoy to Kapica (India). He journeyed through Chighnan and Wakhan. After passing Oudyana, he reached Gandhara, the then Eastern Capital of Kapica. Owing to his illness he failed to return with the envoy and became a monk in one of the monasteries in Gandhara. After a long stay at Kashmir, he returned to Chang-an in 790.

17. CHANG KUANG-YEH 張匡鄴 (938-942).

In 938, during the epoch of the Five Dynasties, Chang Kuang-yeh was appointed envoy to Yu-tien 于闐 (Khotan) by Prince Kao Tsu of Post Tsin 晉高祖. Starting from Ling-chou 靈州 he arrived at his destination after a long journey of two years. He stayed abroad for two years and returned to China in 942.

18. CHI YEH 繼業 (966-976).

In 964-966 T'ai Tsu of the Sung dynasty dispatched 300 monks to India for the purpose of seeking Buddhistic scriptures. Of these Chi Yeh was the best known. His surname was Wang 王. He started from Wutu 武都 district of Kansu and journeyed to Kashmir and Gandhara, after which he reached Jalanda. Then he visited Benares, travelled along the Ganges, whence he proceeded southwards and stayed at Han-shih, 漢寺 one of the many monasteries specially provided for Chinese. Here he remained for several years until 976, when he returned to China.

19. WANG, YEN-TE 王延德 (981-983).

In 981 Wang Yen-te was appointed envoy to Kao-chang 高昌 the Yakoto of today. He started from the Hsia district of Shansi and travelled through Luk-chen 魯克沁. On reaching Kao-chang he discovered a large number of monasteries already established. One of these was in charge of a Persian monk. On hearing that the ruler was at Pechibali 北庭 a summer resort, he proceeded thither. Wang was a celebrated diplomat of the Sung dynasty.

20. CHIU CH'ANG CH'UN 邱長春 A.D. (1208-1288).

Was born at Tengchow 登州 in Shantung. When a boy he was fond of study. At the age of 19 he studied Taoist books and became a priest under the name of Ch'ang Ch'un Tzu 長春子. He was the favourite pupil of Abbot Wang at Ninghai. He was sent for by Jenghis Khan who ordered his personal Minister Liu Wen to invite him wearing a golden tablet in the form of a tiger's head around his neck. On this was written the message: "This man is empowered to act with the same freedom as I myself should exercise, should I come in person."

Chang Ch'un undertook the long journey and eventually reached Samarkand in December, 1221. Early next spring, on May 11, when the almond trees began to bloom, his party of twenty reached the great Khan's camp, who greeted him thus: "Other rulers summoned you, but you would not go to them. And now you have come ten thousand li to see me. I take this as a high compliment." Ch'ang Ch'un replied: "That I, a hermit of the mountains, should come at your Majesty's bidding was the will of Heaven." Jenghis was delighted, begged him to be seated and ordered food to be served. Then he asked him: "Adept, what medicine of Long Life have you brought me from afar?" The priest replied: "I have means of protecting life, but no elixir that will prolong it." The Emperor was pleased with this candour, and had two tents for the visitors put up to the east of his own.

After Jenghis Khan's death Ch'ang Ch'un served Kublai Khan, the first Yuan Emperor of China. He wrote a famous Book of Travels called Hsi Yu Chi 西遊記 or Annals of Western Travels.

Two works have tended to be confused with the present book.

(a) The Hsi Yueh Chi 西域記, describing the pilgrimage to India of the great Buddhist traveller Hsuan-chuang (seventh century).

(b) The Hsi Yu Chi 西遊記 of Wu Ch'eng-en (end of the sixteenth century), a fantastic novel which is to some extent a parody of the *Hsi Yueh Chi* of Hsuan-chuang. For a long time this novel was supposed to be the work of Ch'ang-ch'un, a mistake only possible because both books possessed the same name and till the nineteenth century very little was known either about Ch'ang-ch'un himself or about the book in which his Travels are described.

21. JENGHIS KHAN 成吉思汗 (1162-1227).

A whole book may be written about this mighty warrior of the 12th century, who, without education or influence, fought

his way up as a Mongol chieftain, became a world conqueror and left his name behind as perhaps the greatest militarist who had lived, surpassing even the mighty Napoleon at his height. As a boy already, Temuchin (as Jenghis was then called) had had visions of leadership and conquest, and when the opportunity came, he seized it with both hands, raised immense armies both among his Mongolian hordes and the numberless tribes that he conquered, struck quickly and successfully, and ultimately became the Great Khan of immense territories ranging from China across huge Siberia, Northern India, Bokhara to the borders of the Caspian Sea and Mid-Europe as far as Hungary. Well might Jenghis Khan be named the Mighty Manslayer, the Scourge of God, the Perfect Warrior, and the Master of Thrones and Crowns, as had been given him at various times. A nomad, a hunter and herder of beasts, he outgeneralled the powers of three empires; though a barbarian who had never before seen a city nor known the use of writing, yet he was loved by his adherents from the learned Chinese Minister Liu to his fiercest general, and they served him faithfully until he died in the saddle, in 1227 at the age of 65. No empire of such magnitude stretching over contiguous territory has been known before or since.

22. KUBLAI KHAN (SHIH TSU) 元世祖 (1215-1294).

Kublai Khan, the first acclaimed emperor of the Yuan dynasty, was one of the three grandsons of Jenghis by his fourth son Tule. Kublai and his other two brothers Mangu and Hulagu were all experienced generals; he was given China to subdue, while Mangu had charge of Europe and Hulagu of Persia and Mesopotamia.

From the first, Kublai devoted himself to winning over his Chinese subjects, employed scholars and men of culture, and encouraged the study both of Confucianism and Buddhism. Though a relentless warrior in his younger days, riding hither and thither under forced marches with his invincible troops, he now settled down to days of leisure in a Chinese atmosphere. He transferred his Court from Karakorum to Khanbalig or Cambulac (city of the Khan), as Peking was called in those days, built palaces, became a patron of arts and letters and invited foreign envoys to his country. Hence it was during Kublai's (Shih Tsu's) reign in 1264, that Peking offered hospitality to the first Europeans, and these happened to be two Italians, named Nicolo and Maffeo Polo, who received a warm welcome. Encouraged by this reception, Nicolo on his next visit in 1275 brought his son, Marco, who later through his writings was the means of acquainting a sceptical West with the extraordinary treasures and highly developed civilisation of Cathay. Kublai's reign was a most successful one, for

he was ruler of practically all Asia and the West as far as Scind, Syria and Hungary and also on cordial relations with most foreign countries. Three interesting events occurred during his reign, having a bearing on our subject, namely:

(a) *Wars with Japan*.—The cause of these wars was Kublai's mad desire to receive tribute from Japan. The king of Korea was his son-in-law, and through him Kublai demanded that the Islanders should acknowledge him as overlord. To his dismay he found them as stubborn as he himself was haughty, for they notified him of their unwillingness to comply by the murder of his envoys. In 1274 an armada of 900 vessels conveying 15,000 Korean and 25,000 Tartar soldiers under Gen. Hu Tu 忽都 and Hung Ch'a-chiu 洪茶邱 was defeated at Tsushima 對馬, but this failure only served to stimulate Kublai to greater efforts some seven years later. By 1280 he was in possession of South China, and at the ports of Fukien, rich in timber, ship-building was pushed on with great energy, while recruiting agencies were established throughout the empire. In 1281 a fleet of 4,500 ships, manned by Mongols, Chinese and Koreans under Ou Lu-han 阿樓罕 and Fan Wen-hu 范文虎 sailed proudly in the direction of Hakata. But the resistance offered by the Japanese was so strong that for two months every attempt at landing was frustrated. While cruising fruitlessly in the vicinity of Hichiku, the fleet encountered a severe storm which sent the majority of the ships to the bottom of the sea, leaving a few of the survivors to go home and tell the sad story. Further attempts at revenge were given up because of the unpopularity of the enterprise.

(b). *Expedition Against Champa*.—This also came about through Kublai's demand for tribute. Champa was in that part of the peninsula now called Cochin-China. To invade it necessitated the passage of a Mongol army through Annam, nominally a vassal state of China; but his plan was fiercely contested. The tropical heat proved more deadly than any weapons of the enemy. Stricken down by dysentery and malaria, they soon found it necessary to retire, and it was at this juncture that they were fallen upon by the hidden foe with great slaughter.

(c). *The Great Commercial Routes*.—In spite of these disasters, the Mongols reached the sea. They had three routes by which they communicated with Europe; the two land routes through Nan Lu (Southern) and Pei Lu (Northern), and the maritime one. The last had been the route of the Chinese and the Arabs, and lay along the vast coast line extending from ports in Chekiang and Fukien to Genoa and Florence. Trade was either carried on through the land routes to Kara-korum and Peking, or by sea through the ports of Amoy, Canton, Foochow and Hangchow. While the accidents of war, diplomacy, and

other circumstances brought men in all walks of life from the outside world into China through these routes, the Chinese also found their way into lands far from home. It is stated that about this time Chinese engineers were employed on the banks of the Tigris, and Chinese astrologers and physicians could be consulted at Tabriz.

23. BRIDE OF ILKHAN ARGON (1293).

This was a Mongolian princess, who was chosen as bride when seventeen years old to proceed to Persia to be married to Argon, the Ilkhan (Governor) of Persia. Argon was the grandson of Hulagu, grandson of Jenghis and the first Ilkhan of Persia. It happened that the Polos after twenty years' continued stay in China were anxious to return to Italy and so were invited to accompany the bridal mission by sea. The party sailed from Wenchow in 1291, stayed several months in Sumatra and South India on the way, and by the time they reached Persia, the old Ilkhan had passed away. So the princess married the son, his successor, in 1293.

24. CHANG TE-HUI 張德輝 (1247).

Chang Te-hui was a learned scholar who left Tingchow 定州 in 1247 for Karakorum at the invitation of the Ku-yuk Khan. He stopped over at Peking, Nankow and Dalainor on his way.

25. CHOU TA-KUAN 周達觀 (1296-7).

Since the wonderful ruins of Angkor in ancient Cambodia were excavated by the French authorities, there have been ceaseless attempts to trace their history. Fortunately, through the efforts of M. Pelliot, certain memoirs of a Chinese contemporary named Chou Ta-kuan have been discovered and translated into French. Chou Ta-kuan was not a regular official though attached to the Chinese embassy sent by the Yuan emperor in 1296 to visit the king of Cambodia. In other words, Chou acted in the capacity of unofficial chronicler. Nevertheless, his *Memoirs on the Customs of Cambodia* (Chen La Feng T'u-chi 真臘風土記) have been the principal means in revealing the state of civilization existing in that region during the 13th century. Chou was a keen observer. His book contained thirty-three chapters of useful observation ranging from description of the city walls, style of houses, dress, ceremonies, sickness, burial rites, law enforcement, etc. to commerce, flora, fauna, wines, baths and the manner of living among both princes and the common populace. The writer mentioned that the city wall was 20 li around and 20 feet high, had five gates, with a golden pagoda in the middle and one copper one behind. The king's palace was built of solid carved granite and was most imposing, rising to great heights of architectural splendour. The weather

being ever warm, the inhabitants dressed simply in a one-piece wrap of cloth. The princes lived a life of ease, went out freely with a huge retinue, wore plenty of jewellery and painted the palms of their hands red like the women. Leprosy was prevalent but not regarded with awe, for the patients mixed freely with healthy persons and one king actually suffered from the disease.

The mission left Wenchow by boat in the second month of 1296, reached Champa in the third month, but Cambodia only in the seventh month because of storms. Here the embassy stayed for 11 months and returned to Ningpo in the 8th month of the following year.

26. CHENG HO 鄭和 (1407-1427 out 7 times).

Cheng Ho—native of Yunnan and popularly known as San Pao T'ai-chien 三保太監 was a famous eunuch sent by Emperor Yung Lo on extended mission to the "Western Ocean" to overcome his nephew, the second Ming, whom he had dethroned in 1402.

Conscious of his position as a usurper and fearing that his nephew might have escaped abroad, Young Lo sought to establish himself in the eyes both of his own subjects and of foreigners by a forceful naval diplomacy, which was at the same time materially profitable. He sent out a series of powerfully armed expeditions with Cheng Ho in command to visit the various islands and littoral states of the South China Sea and the Indian Ocean, present gifts at their courts and persuade them to send embassies with tribute to China in return. The following extract is from the Government Chronicle of those days:

His Majesty, under the suspicion that the ex-Emperor Chien Wen might have fled to countries beyond the sea, commissioned Cheng Ho, Wang King-ho, and others, to pursue his traces. Bearing vast amounts of gold and other valuables, and with a force of more than 37,000 soldiers under their command, they built great ships—sixty-two in number—and set sail from Liu Chia-chiang (Liu-ho), in the department of Soochow, whence they proceeded, by way of Fukien to Chen-Cheng, and thence on voyages throughout the western seas. Here they made the knowledge of his majesty and goodness. They bestowed known the manifestoes of the Son of Heaven, and spread abroad gifts upon the kings and rulers, and those who refused submission they overawed by force. Every country became obedient to the Imperial commands; and when Cheng Ho turned homewards, they sent envoys in his train to offer tribute. The Emperor was greatly pleased, and after a short interval commanded Cheng Ho to go once more and distribute gifts among the different States. The number of those who presented them-

selves before the throne grew ever greater. Cheng Ho was commissioned on no less than seven embassies, and thrice he was made prisoner by foreign chiefs. His exploits were such as no eunuch before him, from the days of old, had equalled. At the same time, the different peoples, attracted by the profit of Chinese merchandize enlarged their mutual intercourse for purpose of trade, and there was uninterrupted exchange of commodities. Thus it came to pass that in those days there was the saying of 'The eunuch San-pao who went down to the West;' and all who, in after times, were sent as bearers of commissions to the countries by sea, were wont to impress the outer nations with Cheng Ho's name. Yet, as regards China, the treasure that was lavished in these undertakings brought no profit in return; whilst of the soldiers of the expeditions, many perished by shipwreck or were cast away in distant lands, so that the number who returned, after nearly a score of years had elapsed, was not more than one or two in ten."

These expeditions were primarily pacific in intention, but they carried strong detachments of troops who were available to punish insult or treachery towards the envoys. In Sumatra and Ceylon some hard fighting had to be done by the Chinese armada. In Ceylon King Wijayabahu VI had maltreated a Chinese mission bringing offerings to the Shrine of the Tooth of Buddha at Kandy; in 1410 the Chinese deposed him by force and intrigue, and carried him a captive to China. His successor, invested as a vassal of the Ming, paid regular tribute to China until 1459. To the west of Ceylon the Chinese ambassadorial fleets paid calls as far as East Africa; among the places they visited were Cochin, Quilon, Calicut, Ormuz, and Magadoxo. Both Aden and Magadoxo sent embassies to the Chinese court in 1427.

When Ceylon after 1459 discontinued the payment of tribute, no effort was made to reassert Chinese supremacy; the energy of an earlier generation had dwindled and the attention of the Chinese government was otherwise engaged. The rise of the Kalmuk to power in Central Asia and the renewal of the nomad menace on the northern frontiers had diverted the thought of Chinese statesmanship from remote sea avenues; the Ming capital had been transferred in 1421 from Nanking to Peking, and maritime affairs came to be more and more neglected, the eye of state turning foremost to Mongolia and Manchuria. Yung Lo's forward policy in the Indian Ocean was abandoned, and, lacking official support and encouragement, Chinese private traders also beat a retreat; by the second half of the fifteenth century they had practically ceased to sail west of the Straits of Malacca.

27. CH'IEN LUNG 乾隆 (and HSIANG FEI) 香妃 (1712-1798).

Ch'ien Lung, fourth son of Yung Cheng, ascended the Manchu throne in 1735 and reigned for 60 years. Unlike most emperors he was a great traveller, and numberless stories have been handed down regarding his exploits. Some writers go so far as to say that Ch'ien Lung was really the son of Minister Ch'en, favorite counsellor of the previous emperor, and was surreptitiously exchanged at birth for the baby daughter of Yung Cheng's consort. This might account for Ch'ien Lung's unusual love for art and literature as well as his remarkable penmanship. It might also explain his extensive visits to Hangchow and other southern cities closely associated with the Ch'en household. During his reign, large tracts of the north-west (including Mongolia), Tibet, Nepal, Burmah and Cochin were added to the Chinese Empire. Ch'ien Lung also directed a military campaign to Sungaria (Eastern Turkestan) during which the Mahomedan prince was slain and his beautiful wife captured and brought to Peking to serve as imperial concubine. But this 'fragrant princess' (Hsiang-fei) 香妃 would not consent and for years lingered in the Forbidden Palace with a dagger hidden in her bosom ready to kill the emperor and then herself should the former force his attentions upon her. Ch'ien Lung was madly in love with her, granted her all sorts of privileges and erected a special mosque on the other side of the avenue facing the southern tower, so that she might see her people and even worship with them according to Mahomedan rites. After all attempts failed, the aged mother of the emperor summoned the obstinate princess and demanded to know what her intentions were. "I would rather kill myself than forget my dead lord's memory and give in to your son," said the young widow. "In that case, take this cord and carry out your intentions quickly," ordered the dowager. So she died. When Ch'ien Lung heard of the incident, he wept long and earnestly, and conferred the posthumous title of 'senior concubine' upon Hsiang Fei.

28. HUANG K'UAN (WONG FUN) 黃寬 (1828-78).

This was the first Chinese to have obtained a medical degree from any European or American college. Wong Fun was one of three Cantonese (the other two being Yung Wing and Wong Shing) who accompanied an American missionary Mr. R. S. Brown in 1844 to America for study. While his companions remained in America, Wong Fun proceeded to Scotland and finally took his medical degree in 1853 at Edinburgh University. He returned to Hongkong and practised. He was also the first Chinese medical officer of the Customs, and in the capacity of a surgeon earned a considerable fortune. He never married.

29. LI HUNG-CHANG 李鴻章 (1822-1901).

Was a learned Hanlin scholar, born in Anhui, who, through his association with Marquis Tseng Kuo-fan in the suppression

of the Taiping rebels (1851-65), rose step by step in office until he became Viceroy of the two Kwang provinces, and later of Chihli. When the Sino-Japanese war broke out in 1894, Viceroy Li was appointed commander-in-chief of the Chinese army and navy. The War ended in the victory of Japan. Li was then sent as envoy to Japan to sign a Treaty of Peace. While there he was shot by a Japanese lunatic, but was mercifully saved. In 1896, Li was commissioned to travel to European countries and introduce reforms for the improvement of China. Both he and his staff learnt much from their world tour, during which they met most of the notabilities of the time like Bismarck, Salisbury, Gladstone, Edison, Maxim, Witte, etc. Viceroy Li was also the founder of the China Merchants Steamship Company and of the first modern government medical college in Tientsin.

30. YUNG WING MISSION 容閔 (1872-1876).

Yung Wing was one of the first three Chinese young men who accompanied Rev. R. S. Brown to America for study. On his return in 1855 after graduation at Yale, he worked energetically for the dispatch of more students to that country. It was not until 1871 that the Imperial government sanctioned an appropriation of one and a half million dollars for the modern training of 120 young Chinese in various American institutions. This was known as the Yung Wing Mission. Unfortunately, when Yung Wing was promoted as associated Minister to Washington, and another official—a strong conservative—became educational commissioner, the latter recommended the suspension of the Mission because the young students appeared to him to have become denationalised and thus a danger to the empire. No more students were sent after 1876, but quite a number of those who returned occupied high official positions afterwards, such as, Liang Yun-yen, Sir Chentung Liang Cheng, Sir Shousen Chou, Liang Munting, Admirals Wu Ying-chi and Tsai Ting-kan, Jome Tien-yu, etc.

31. KANG YU-WEI 康有爲 (1856-1928).

Was the noted reformer and adviser of Emperor Kuang Hsu during the years 1897-98, when the then youthful emperor tried to save his empire from disruption by adopting fundamental reforms in education and government after the shameful defeat of big China by tiny Japan in the war of 1894-95. The empress dowager Tsu-Tsi, however, opposed their radicalism, made a *coup d'etat*, put the emperor in prison, and executed as many of the Cantonese advisers as could be found. Kang Yu-wei managed to escape in a British cruiser from Tientsin, and for years afterwards lived the life of an exile, visiting practically every country of the world to preach the doctrine of constitutional monarchy for the salvation of China. In 1911

the Manchu dynasty fell, but instead of a constitutional monarchy the people declared for a republic. Kang was thenceforth left alone.

32. SUN WEN (SUN YAT-SEN) 孫文 (1866-1925).

Father of the Chinese Republic was born at the Village of Tsuiheng between Canton and Macao. After spending his boyhood in Honolulu proceeded to Hongkong and obtained a licentiate in medicine in 1892 at the Medical College there. From the beginning Dr. Sun was more interested in revolution than in medicine, and spared no effort to achieve the downfall of the Manchus. After the abortive rising of 1896 in Canton, Dr. Sun went into hiding, but his name came prominently forward the following October, when it was announced that the Chinese Legation in London had kidnapped him and was smuggling him back to China in a wooden case. Thanks to the timely intervention of the late Dr. James Cantlie and Lord Salisbury, Dr. Sun was released. He travelled widely, forming secret revolutionary branches among Chinese communities everywhere. When the Manchus abdicated in 1911, Dr. Sun was elected first President of China, but the country has seldom enjoyed peace since then. Dr. Sun more than once barely escaped with his life during these frequent troubles. He died in Peiping in 1925, leaving the famous San Min Chu-I (Three Principles) for the guidance of New China.

33. CH'ENG PI-KUANG 程璧光 (1860-1921).

Was born in Canton and studied for the navy at the Foo-chow arsenal. He fought in the Sino-Japanese War of 1894-5, when his ship was sunk, and he was picked up from the sea. In 1896-98 Captain Ch'eng was in England helping to superintend the building of three cruisers for China. During the later days of the Manchu, he was promoted Admiral and Minister of Navy. It was at this time, 1909-10, that Admiral Ch'eng was ordered by the Throne to visit Mexico with his fleet and protect Chinese traders who had been maltreated in that country. The Mexican Government apologised and promised to behave better in future. For the first time in Chinese history a Chinese fleet steamed round the globe and showed itself to widely-scattered compatriots. When the Republic was proclaimed, Admiral Ch'eng was appointed governor of Kwangtung Province in 1920, but a few months afterwards he was shot by an assassin as he was stepping into a boat to cross the river. Admiral Ch'eng was a man of integrity and one of the finest products of western education.

34. CHIN YAMEI (YAMEI KIN) 金亞梅 (1864-1934).

Is the first Chinese woman to have obtained a medical qualification from the west. She was the daughter of a Ningpo

pastor and adopted by the Rev. and Mrs. McCartee, American missionaries in China. With them the Chinese girl travelled widely and stayed some years in Japan as well as in America. In the latter country she studied medicine at New York and finally graduated first on the list from the New York Women's College of Medicine and Surgery. She was much interested in histological work, her paper on "Photographical studies of histological sections" in the New York Medical JI. being the first scientific article ever published by a Chinese woman. Dr. Yamei Kin died at Peiping at the age of 71.

35. LIN WEN-CHING (LIM BOON-KENG) 林文慶 (1869-)

Born of Amoy parents in Singapore is therefore an Oversea Chinese. He won the first government scholarship of his year in 1887 and was sent to Edinburgh University, where he was awarded high honors in medicine, as well as the degrees of M.B., C.M. Invited by Prof. C. Roy to work in Cambridge, he undertook some research in that university, and in 1892 had the rare privilege of seeing his paper on "Protective Mechanism in *Ascaris Lumbricoides*" published in the Proceedings of the Royal Society. Dr. Lin was adviser on Public Health in the first Cabinet of Dr. Sun Wen in Nanking 1912. Since 1911 he has been President of the University of Amoy.

In conclusion, a few words may be devoted to the tens of thousands of emigrants, who had in the past left the shores of China, mainly from Kwangtung and Fukien Provinces, to help other countries to colonise and thus consolidate their possessions. Their activities include the development of tin-mining and rubber cultivation in Malaya and Netherlands East Indies; the introduction of the sugar industry into the Philippines as far back as the Yuan era, British West Indies and even Hawaii; the building of those immense railway trunk-lines across Canada and America; and the marked transformation of immense areas of arid territory in California, Australia and Mexico into fertile fruit orchards. Wherever Chinese have travelled and settled down, they and their descendants have brought with them the well-tried benefits of their ancient philosophy, culture and industry.

The Chinese language, like English, may perhaps claim to be the most widely used in the world. Now and then, as in the case of a certain family settled down in Trinidad, a genius has blossomed forth from humble emigrant stock to arouse the world with his fiery and eloquent pen and to challenge the wisdom of a placid community.

Chinese travellers have not lived in vain, and their achievements are written large on the face of the globe.

LIST OF EARLY CHINESE TRAVELLERS.

		DATE	DYNASTY
1. Meng Ch'iang	孟 姜	B.C. 500	Eastern Chou (W)
2. Hsu Shih	徐 市	" 220-210	Ch'in
3. Chang Chien	張 騫	" 138-127 (away)	Han
4. Wang Chao-chun	王昭君	" 40	"
5. Pan Ch'ao	班 超	A.D. (32-102)	" (W)
(and Kan Ying)	甘 英	" 87	
6. Chu Shih-hsing	朱士行	" 260	
7. Fa-hsien	法 顯	" 399-414 (away)	Eastern Tsin
8. Sung Yun	宋 雲	" 518	Northern Wei
9. Hui Sheng	惠 生	" 518	"
10. Pei Chu	裴 矩	" 605	Sui
11. Wei Chieh	韋 節	" 605	"
12. Tu Hsing-man	杜行滿	" (602-664)	"
13. Hsuan Chuang	玄 奘	" 629-645 (away)	Tang
14. Wang Hsuan-t'se	王玄策	" 646-661	"
15. I-ching	義 淨	" 671-695	"
16. Wu K'ung	悟 空	" 751	"
17. Chang Kuang-yeh	張匡鄴	" 938-942	Post Tsin
18. Chiyeh	繼 業	" 966-976	Sung
19. Wang Yen-te	王延德	" 981-983	"
20. Chiu Chang Chun	邱處機	" (1208-1288)	Southern Sung
(Ch'u-chi)			
21. Genghis Khan	成吉思汗	" (1162-1227)	Yuan
22. Kublai Khan	忽必烈	" (1215-1294)	"
(Shih Tsu)	汗世祖		
23. Bride of Ukham		" 1280	" (W)
Arghun			
24. Chang Te-hui	張德輝	" 1247	"
25. Chou Ta-kuan	周達觀	" 1296	"
26. Cheng Ho	鄭 和	" 1407-1427 (out 7 times)	Ming
27. Ch'ien Lung	乾 隆	" (1712-1798)	Ching (W)
(and Hsiang-fei)	(香妃)		
28. Huang K'uan	黃 寬	" (1828-1878)	"
(Wong Fun)			
29. Li Hung Chang	李鴻章	" (1822-1901)	"
30. Yung Wing Mission	容 閔	" (1872-1876)	"
31. Kang Yu-wei	康有為	" (1856-1928)	"
32. Sun Wen	孫 文	" (1866-1925)	"
33. Cheng Pi-kuang	程璧光	" (1860-1922)	"
34. Yamei Kin	金亞梅	" (1864-1934)	" (W)
35. Lin Wen-ching	林文慶	" (1869-)	"

Figures inside brackets—span of life
Ordinary figures—years of activity.

APPENDIX

WU LIEN-TEH—A SHORT AUTOBIOGRAPHY

At fifty years of age one looks back upon the past with a rather detached mind. One's early ambitions are somewhat toned down, criticisms of one's fellow creatures become fewer and less severe, and while surveying present-day conditions one tends to view things in a more optimistic spirit than in the earlier years.

Hence, in writing this short autobiography I would ask my readers to excuse me if I miss out the unessential parts and devote the following pages to persons, institutions and surroundings I have been associated with in the manner of a moving picture screen, leaving to my readers any deductions to be gathered in whatever way they consider fit.

I was born in 1879 in the Island of Penang, Straits Settlements, that stretch of British territory formerly belonging to the Malays and now intensely prosperous as a result of the tin and rubber industries. My father was of the usual Cantonese emigrant type who left his village home in Sinning (now Tai-shan) to make his fortune across the "Seven Seas." My mother belonged to the second generation of Chinese in Malaya, her father being an emigrant but her mother was descended from resident Hakka parents, those sturdy fearless peasants of China who though possessing no province of their own are nevertheless able to succeed in business where others fail. Until within recent years the education given by the local government was purely in the English language. The fees were low, varying from six to twelve dollars a year, so that even the poorest children could take advantage of it. I stayed in the Penang Free School for nearly ten years (1886-1896) and learnt not only how to write and read English but also a lot about English History and Literature, English Geography (including details of the smallest hamlet), Latin, Chemistry and Geometry. I won many prizes at school, but the most difficult to obtain, namely, the Queen's Scholarship, for which candidates had to proceed to Singapore to be examined, required three attempts before I was successful. On the first two occasions I was under-age, but in 1896 I reached the goal of my boy's ambition and was awarded the much coveted prize of that year, thus enabling me to proceed to England and enter my name as an undergraduate of science and medicine at Emmanuel College, Cambridge. The

value of this scholarship was £200 per annum, out of which income tax had to be deducted by the London Government. Those who are conversant with university life in England will understand that this sum, even in those days, was barely sufficient to carry a student through the year, especially as travelling expenses, clothing, college fees, and holidays were included therein. However, by exercising the strictest economy and avoiding luxuries of every sort I managed to keep my head above water. The first year was the hardest, for the fees were then the highest and mistakes were frequent. Fortunately I had a sympathetic tutor at College in the person of Dr. William Napier Shaw (now Sir W. N. Shaw, Head of the Meteorological Office, London) who did all he could to encourage me in my studies and aspirations. At the end of my second year (June 1898) I won a College Exhibition of £40, which enhanced my slender income and enabled me to partially return the hospitality which I had earlier received from my friends. The next year (1899) brought still better luck, for I obtained First Class Honours in the Natural Sciences Tripos for the B. A. and was rewarded with a Foundation Scholarship of £50. In addition I passed all the necessary examinations for the second M. B. Examination in the shortest possible time.

In September, 1899, I removed to London, competed for and won the University Scholarship at St. Mary's Hospital open to graduates of British Universities. The money from this new prize just covered all tuition fees required during the next three years at the Hospital. I found student life in London quite tame and unexciting after the "jolly time" at the 'Varsity. Gone were the four o'clock teas, evening squashes, May Weeks, Bumping Boat races, noisy rags and wooden spoons. In their place I had to be satisfied with attendance at Casualty, Clinical clerkships, surgical dressing, midwifery cases among the London poor, witnessing post mortems and all sorts of surgical operations. But the change was good for it sobered one to the serious problems of life and opened one's eyes to the vastness and complexities of human toil. From that time medicine appealed to me more than ever, and its numerous possibilities in China were rendered more and more evident.

After two and a half years in the British metropolis, I qualified as a Bachelor of Medicine and Bachelor of Surgery (M.B., B.C.) of Cambridge in April, 1902, being the only medical student of that University out of a class of 135 who entered in 1896 to have accomplished that task in the short period of $5\frac{1}{4}$ years. I also won the Cleadle Gold Medal in Clinical Medicine and the Kerslake Scholarship in Pathology and Bacteriology, besides various prizes in surgery and medicine. The next problem was to increase one's experience, for it has been truly said

that 'a man only begins to learn after obtaining his degree.' My old College (Emmanuel) offered me a Research studentship of £150 a year for 1902-3 to carry on scientific investigations in various laboratories. This opportunity I readily seized and spent the ensuing year working first under Dr. Ronald Ross (now Sir Ronald Ross) at the School of Tropical Medicine, Liverpool, then under Professor Karl Fraenkel at Halle a/S in Germany, and lastly under Professor Elie Metchnikoff at the Pasteur Institute, Paris. I devoted my time principally to research upon malaria and tetanus, and thanks to association with prominent thinkers in the world of Science I managed to imbibe its true spirit and to hope for the time when I might pass on the same to others who came after me. Living among continental people and learning their languages enabled one to become simpler in habits, more tolerant and broadminded. I acquired a fair knowledge of German and French without a teacher by mixing freely among the people and sharing their tastes and enjoyments, however humble these might be.

On my return to England in the spring of 1903 I was advised by the then Regius Professor of Physic of Cambridge (Sir Thomas Clifford Allbutt)—most kind-hearted and brilliant man—to utilize the results of my investigation on tetanus for my M. D. thesis. This examination I successfully passed that year, though I had to wait another two before the degree was actually conferred upon me. I also accepted the post of House Physician at Brompton Hospital for Consumption and Diseases of the Chest, London, for six months, where I gained an insight into the wide prevalence of Tuberculosis and its effective control.

I returned to the Straits at the end of 1903 with another year of the College Fellowship and joined the Institute for Medical Research, Kuala Lumpur, for the study of Beri-Beri, which disease killed thousands of Chinese and Indians annually in those regions. The next three years (1904-07) were spent upon private practice in Penang, but although my bank balances increased day by day, my heart always yearned for a chance to use my unusual experience for higher purposes than the mere treatment of common ailments so often associated with general practice. The declaration in the British Parliament of Mr. John Morley (then Secretary of State for India) in 1906 that the Indo-Chinese opium trade was an immoral and indefensible one had given new life to anti-opium workers, and I entered the fray with the greatest enthusiasm, helping my friends to subscribe tens of thousands of dollars for the cause, establishing homes for addicts and giving free medical services to them. Unfortunately this work conflicted with the aims of several rich families whose source of income was mainly derived from the opium monopoly and resulted in much opposition and abuse from

both officials and would-be-patients. However, the campaign against the opium habit rapidly spread and encouraged fellow-workers in China and elsewhere to persist in their efforts. The year 1907-8 could certainly be designated a red letter day in anti-opium history. In August 1905 I married Miss Huang Shu Chiung, second daughter of Mr. Huang Nai Siong of Foochow. We first met one another at the home of Dr. Lin Wen Ching, a leading practitioner of Singapore, whose wife was Mrs. Wu's sister. Our married life has been an unusually happy one, and our only son Chang Keng is now studying medicine at John Hopkins University, U.S.A.

In July 1907 I paid my first visit to China and visited Tientsin, where I was urged by Viceroy Yuan-Shih-Kai (as he then was) to join the Chinese Army Medical Service. At the critical moment I was attacked by acute dysentery, which necessitated my leaving the north for a time, though it also made possible my visit to military institutions in London and Berlin for further experience. Next year 1908 when I arrived at Peking, both the Empress Dowager and Emperor Kwang-Hsu had died, Yuan-Shih-Kai had been banished to Honan, and his principal enemy, the Manchu Tieh-Liang, was in charge of the Board of War. However, through the influence of my old friend Admiral Tan Hsueh Heng, I was appointed to the post of Vice-Director of the Army Medical College, Tientsin, where young men were being prepared as army medical officers for various units of the modern army. The teaching was done in the Japanese language by Japanese and Japanese-trained Chinese, and insufficient emphasis was laid upon practical work and the prevention of diseases.

In December 1910 came news of the outbreak of pneumonic plague in Manchuria, where the local officials and old-style physicians were helpless in the presence of the rapidly growing number of victims. Political complications were also feared because Russia and Japan had both threatened to send their own medical staff and military into the infected areas unless more radical measures were adopted by the Chinese. Mr. Alfred Sze (then Councillor of the Foreign Office) sought me out and asked me to proceed immediately to Harbin with another Chinese doctor connected with the Board of Navy. I readily agreed and within two days after receiving instructions was off. My colleague did not come and so missed a fine opportunity.

The campaign against the Manchurian Plague Epidemic of 1910-11 is pretty well known to most readers of modern history and need not be mentioned in detail here. From a scientific standpoint this dreadful disease (which killed almost 100% of those attacked) offered unrivalled opportunities for research, for up to then very little had been known of the infection and

much initiative as well as courage was required for its prevention and treatment. I acted virtually as Commander-in-Chief of the huge organisation and gave orders to doctors, police, military and civil officials alike. The most dramatic phase of that historical fight was when I boldly asked for Imperial Sanction to cremate three thousand odd plague corpses which had for weeks been lying unburied on the ground because of the lack of Coolies and frozen state of the ground. Nearly one-third of the whole population had either died or run away. I waited anxiously for 48 hours for news from Peking, and when permission was finally granted it took only two days to dispose of the dead. After that the medical staff and assistants breathed easier and worked with a more willing heart. The last case in Harbin was recorded on March 1st, that is four weeks after Imperial Sanction was received for cremation. The plague of 1910-11 spread to Chihli and Shantung (including Tientsin, Peking, Tsinan, Chefoo, etc.) and claimed altogether 60,000 deaths.

The successful control of this epidemic considerably affected my fortunes, for together with H. E. Alfred Sze I was commanded by the Imperial Government to organise the Mukden Plague Conference (April, 1911), at which experts from 11 leading nations participated. I sat throughout as Chairman of the Conference and with the other Delegates recommended the establishment of the present North Manchurian Plague Prevention Service to study the disease and cope with future outbreaks. The high rank of Major of the Imperial Army with blue Button was conferred upon me overnight so as to enable me to receive Imperial audience without unnecessary formalities. It seemed as if modern medical science, which in China used to be compared to a barber's profession and had stood under a cloud for centuries, had at last come into its own. Its future prospects were indeed bright. I was also awarded the degree of Chin-Shih or Litt. D. of the Imperial University without the usual examination. The Czar of Russia conferred upon me the Order of Stanislaus Second Order, while the French Government gave me the coveted distinction of Legion d'Honneur.

The Manchurian Plague Prevention Service was started in 1912 with initial funds totalling \$180,000 for buildings and equipment provided by Viceroy Chao Erh Sun of Manchuria, while the annual appropriation of \$120,000 for maintenance is defrayed by the Maritime Customs. Hospitals are established at Harbin (Headquarters with Central Library and Research Institute), Manchouli, Sansing, Lahasusu, Taheiho and Newchwang. There are affiliated hospitals at Antung, Hailar and Tsitsikar. Besides accommodation for plague patients the Manchurian Plague Service treats other communicable diseases like cholera, smallpox, scarlet fever, typhus, etc., carries on routine

hospital work of the city by treating medical and surgical cases, manufactures sera and vaccines for plague, cholera, rabies, scarlet fever, etc., performs chemical and bacteriological analyses on food stuffs, water, patent medicines, etc., and advises the local municipal administrations on health matters.

The effects of this Service have been far reaching, for since 1912, post mortems were officially permitted in 1913, the National Medical Association of China with a membership of 500-600 was established in 1914, western medicine was officially recognised by the Central Government in 1915, and the Central Epidemic Bureau (Peking) was established in 1919.

Returning to the personal factor, I was appointed one of the three Imperial Delegates to attend the Hague Opium Conference in 1911 and signed with the representatives from other countries the historical document known as the Hague Opium Convention of 1912. Next year I visited Europe again and joined His Excellency W. W. Yen (later Prime Minister) as delegate of the second Hague Opium Conference. When the Revolution came in 1911 and China became a Republic I received the appointment of Physician Extraordinary to President Yuan Shih-Kai. This appointment has been continued by successive presidents. In 1915 I proceeded to Hongkong to accept the honorary degree of Doctor of Laws (LL.D.) of the University. On that occasion I took the opportunity of pleading for a more liberal attitude in the appointment of promising Chinese doctors to various teaching posts in the University, and emphasised that besides teaching and the granting of degrees, a University had other functions, such as, research and training of teachers for the hinterland, both of which could best be done by natives of the soil. I am glad to notice that the Professorship of Pathology has since that date been occupied by a Chinese, and that more Chinese have been appointed to responsible posts. Incidentally the second Conference or National Medical Association with me as President was held at Canton in 1916 side by side with the China Missionary Medical Association. At that meeting I urged my colleagues, especially those in charge of medical colleges, to emphasize the needs of public health in their curriculum, for the results so far as actual saving of lives was concerned would far exceed the benefits derived from mere treatment.

In 1916 Mr. Chou Hsueh Hsi (son of the famous Viceroy Chou Fu of Liangkwan) was Minister of Finance under President Yuan Shih-Kai. He asked me to visit him at the Western Hills, Peking, and help him to build a Sanatorium for tuberculous patients at that lovely spot surrounded by 200-400 year-old cypresses and silver-pines, many of them planted

under the supervision of Emperor Chien-Lung who used the place as a hunting park. Having considered the matter I made bold to suggest the erection of a model general hospital in the heart of the capital rather than a restricted sanatorium in the country, however well equipped. He concurred with my views and called a meeting of the leading officials and philanthropists of Peking to discuss the matter, promising to place the 120,000 dollars which he had saved from the salt revenue at the disposal of the Committee. Thus was born the nucleus of the Peking Central Hospital of which I was soon elected Organiser and Medical Director. The task imposed upon me was unusually difficult, for I had to interview influential persons for subscriptions as well as consult architects regarding plans, supervise the complicated ferro-concrete building as it grew up, order the thousand and one articles of equipment, and see to their transportation at the least possible expense, with duty free, half-rate on railways, special discount from firms, etc. In this work of preparation I had the close cooperation of Mr. Sze Sao-Tseng (brother of Minister Alfred Sze of Washington). The Central Hospital costing just over \$300,000 was finally opened in January 1918 amid universal approval, for it was then the finest and most up-to-date institution of its kind in China. Even the Director of the Dairen Hospital (South Manchurian Railway) asked me for a copy of our plans in order that he might use them for modelling the new hospital at Dairen. This latter institution was completed in 1926 at a cost of over seven million yen.

Towards the end of 1917 Pneumonic Plague again invaded China, this time from Inner Mongolia by way of Patsebolong, Suiyuan and Tatung (Shansi). Along with two American volunteer missionary doctors we made our headquarters at Fengchen (Shansi) but the local officials and inhabitants were obstinate, and the special car in which we lived was one day surrounded by a mob and nearly burnt. Other doctors later arrived and the epidemic dragged on, though the region through which it traversed was a sparsely populated one, and some cases reached Peking and Nanking. The total death roll was 16,000, the last case being reported at the end of May (seven months after the commencement of the outbreak).

We went through a serious cholera epidemic in Manchuria in 1919, followed the next winter by the second Manchurian Plague outbreak. Fortunately, on this occasion we were well organised for fighting the pest, which claimed only 8,000 victims instead of 60,000 as in 1910-11, although the population of Manchuria had doubled during the intervening ten years. Our staff was also able to undertake valuable research work, and our reports contributed considerably to the advancement

of knowledge on this important fatal disease. The world now accepts our contention that the Mongolian marmot (tarabagan), whose size is that of a cat and whose thick fur is much valued, is the main reservoir of the germ causing pneumonic plague and that simple handling of the sick animal or freshly dissected skin by the hunter is liable to pass on the infection to him. The insanitary underground inns where these hunters live in winter to escape the severe cold contribute largely to the spread of an epidemic. Instead of entire prohibition of hunting, we have introduced laws for its regulation and have taught the hunters to detect and avoid sick animals, to be vaccinated against plague, and to have the animal skins stored and disinfected at our stations at Manchouli and Hailar. We are confident that although there will always be sick tarabagans, human infections will become rarer and rarer, and even when these do occur widespread epidemics like those of 1910-11 and 1920-21 will be things of the past.

In 1922, I received the honorary degree of Doctor of Science (Sc. D.) from St. John's University, Shanghai. In 1923 I was asked by Marshal Chang Tso Lin of Manchuria to build the North-Eastern Hospital at Mukden capable of accommodating 500 patients. This new structure was completed in June 1924, and consists of a Central Administration Block with covered pathways leading to eighteen separate verandah units, each with its own Arcola heating system and lavatory arrangements. A railway platform reaching the gate enables wounded patients to be conveyed to the wards without undue exertion. The total cost of this new Hospital is \$700,000.

In August 1924 I obtained a Fellowship of the International Health Board of the Rockefeller Foundation to study modern health work in America and spent the greater part of a year at the Johns Hopkins School of Hygiene where the C.P.H. was granted me. I also visited other health and research centers, and brought back new methods for the protection of children against scarlet fever, which malady was particularly virulent in the north of China. For nearly two years now we have conducted this new line of investigation and have obtained very encouraging results.

My 'Treatise on Pneumonic Plague,' upon which I had been engaged ever since 1911, was at last finished in 1926 and was submitted as a Thesis to the Imperial University of Tokio. For this work the rare honour of *igaku-hakushi* (Tokio) was conferred upon me, such a degree not having previously been given to any non-Japanese, Chinese or European. The League of Nations, Geneva, published this book in November, 1926.

Early in 1927 I was invited by the Health Section of the League of Nations to visit certain health centers and research

institutions in Europe. I accordingly left Harbin on March 1, travelled *via* the Siberian Railway, visited in succession Warsaw, Vienna, Berne, Geneva, Lubliana, Zagreb, Belgrade, Paris, Havre, London, Cambride, Hamburg and Berlin, before returning by the same route. The whole trip covered just four months, but as I had been to most of these places in previous years and could speak French and German besides English I managed to see more than the average traveller in such a short time. I was indeed impressed by the extraordinary changes that had taken place since the Great War. Instead of despondency in the defeated countries there prevailed the greatest determination among all classes to make the best of the situation and to adapt themselves to the fresh needs of the times. The victor countries, on the other hand, received additional stimulus and utilised every effort to come up to the standard of America in regard to efficiency and mass production. Education has become more practical and business methods have been made less wasteful. Even medical training is becoming more preventive, so that the fewer births now taking place may live healthier and longer and cost the country less through sickness. A new world is being born!

I proceeded to Calcutta (India) in December 1927 to attend with two other Chinese colleagues the seventh Congress of the Far Eastern Association of Tropical Medicine and for the first time had the pleasure of meeting the leaders of medicine in India. In spite of the forty-century old civilisation of that vast Empire and the consequent conservatism and religious bigotry of the masses, large numbers of Indian doctors have distinguished themselves in the field of Ophthalmology, Surgery, Inner Medicine, Pharmacology, Bacteriology, etc., in addition to Biology, Physics and Mathematics. The names of Bose, Raman. Row and Chopra are as familiar in the west as in the east. But the application of modern health knowledge to the daily life of the average peasant and toiler is still much needed, and strenuous efforts are required in every direction to guide the ignorant populace in the right direction. The same difficulties which we have encountered in China are being experienced in India, perhaps to a greater degree.

My three months tour in India, during which I visited north and south as well as east and west and travelled over 12,000 miles, will long live in my memory.

As a result of the establishment of a National Government in Nanking, the Ministry of Health was inaugurated in October 1928, with Mr. Hsueh Tupi and later on Dr. Liu Juiheng (M.D. Harv.) as Minister. Under the latter's direction, much progress has been made in placing medical and health matters upon a truly scientific foundation. Chief among the

achievements of the new Ministry are: (a) close and permanent co-operation with the Health Section of the League of Nations, whose Director (Dr. Ludwig Rajchman) has already paid two extended visits (1929 and 1930-1) to China and established at least ten travelling scholarships for promising Chinese doctors to study special health and quarantine matters in Europe and America; (b) the coming-into-being of the Central Field Health Station in Nanking, containing special departments of medical activities for training future leaders for various provinces to work along definite time-saving lines; (c) the starting of an independent Quarantine Service with myself as Director-General (for this purpose, the ports of Shanghai and Amoy have been taken over from the Customs with Swatow to follow in April; (d) the registration of medical practitioners, pharmacists and nurses.

I have since the beginning of 1930 been appointed Chief Technical Expert of the Ministry of Health to advise the Government on important health matters. The future of medical work in China certainly looks much rosier than it has ever done, and with more and better trained graduates added to the list as years go by, we shall indeed make considerable headway.

Now I have reached the end of my autobiography. I have left out the part I took in founding and editing the National Medical Journal, my literary contributions to various medical and scientific journals in China, America, Europe and Japan, my lecture tours in Japan, Europe, Java, Malaya, India and Burmah, my attendances at medical congresses in London, America, China, India and Japan, and my co-operation with a long list of friends (Chinese and foreign) in the work of the Y.M.C.A., Y.W.C.A., Council of Health Education, National and International Anti-opium Associations, Medical Schools scattered in different parts of China, etc.

For although much has been accomplished, still more has to be done. I verily believe that China, with her many-thousand-year-old civilisation, industrious peasants and brilliant thinkers and artistic scholars, as well as inherent democratic spirit, can easily get into line with modern nations. But in order to achieve the quickest and most permanent results in the most economical way, her leaders should absorb the best that the West can offer, such as, seriousness of purpose, service to others as well as self, a scientific temperament, rigid scrupulousness in the management of business undertakings, attention to details and a willingness to learn from outsiders even at the height of success. On the other hand, they should

eschew the weaker point of western civilisation, such as, undue worship of material success at the expense of the soul, over-indulgence in the ordinary comforts of life, numberless luxuries and lack of discipline in the family.

WU LIEN-TEH.

Shanghai, Jan. 1931





Delegates to the International Plague Conference, Mukden, 1911.

<i>First Row:</i> —Dr. Broquet (France)	<i>Second Row:</i> —Dr. Haffkine (China)	<i>Third Row:</i> —Dr. Petrie (Great Britain)
Dr. Galeotti (Italy)	Dr. Gray (Great Britain)	Dr. Di Giura (Italy)
Dr. Strong (America)	Dr. Farrar (Great Britain)	Dr. Signorelli (Italy)
Dr. Zabolotny (Russia)	Dr. Fang (China)	Dr. Koulecha (Russia)
Dr. Kitasato (Japan)	Dr. Fujinami (Japan)	Dr. Stanley (China)
Dr. Wu Lien Teh (China)	Dr. Aspland (China)	Dr. Hill (China)
<i>President</i>	<i>Medical Secretary</i>	Dr. Wang (China)
Dr. Martini (Germany)	Dr. Christie (China)	Dr. Chabaneix (Deputy)
Dr. Worell (Austria Hungary)	Dr. Chuan (China)	
Dr. Shibayama (Japan)	Dr. Shimose (Japan)	
	Dr. Teague (America)	



Delegates of the International Plague Conference in Session, Mukden, April 1911. Dr. Wu Lien-teh (President) with Prof. S. Kitasato on his left.



Picture taken in 1911 of vacant site in Harbin before erection of various buildings of the Manchurian Plague Prevention Service. In background were wooden sheds for housing plague contacts. In foreground are (left to right) police commissioner Moh Teh-hui (later special ambassador to U. S. S. R.) ; taotai Li Chia-ao and magistrate Lin Shou-t'ing.



Anti-Plague Institute, Harbin (Manchurian Plague Prevention Service). Building at the left is the General Hospital, built in 1922; in the centre is the Administration Block, erected 1920; in front is the Laboratory and Institute Block completed in 1924.



Winter view of the East or Infectious compound, Harbin Anti-Plague Institute.



Arctomys bobac, the Siberian marmot or Mongolian tarabagan, size of an adult cat.



Skull of *Arctomys bobac*, posterior and lateral views.



Wild rodents of Manchuria and Siberia:

1. *Arctomys bobac* (Tarabagan).
2. *Citellus mongolicus* (Suslik).
3. *Dipus sowerbyi* (Spring hare).
4. *Meriones kuraochii* (Spring mouse).
5. *Siphneus aspalax* (Grey mole rat).
6. *Micromys agrarius* (Striped field mouse).



Piles of coffins ready for cremation (Manchurian Pneumonic Plague Epidemic of 1911).



Inside Compound of Harbin Plague Hospital, 1921 epidemic.



Type of mask worn in the pneumonic plague epidemics, Manchuria.



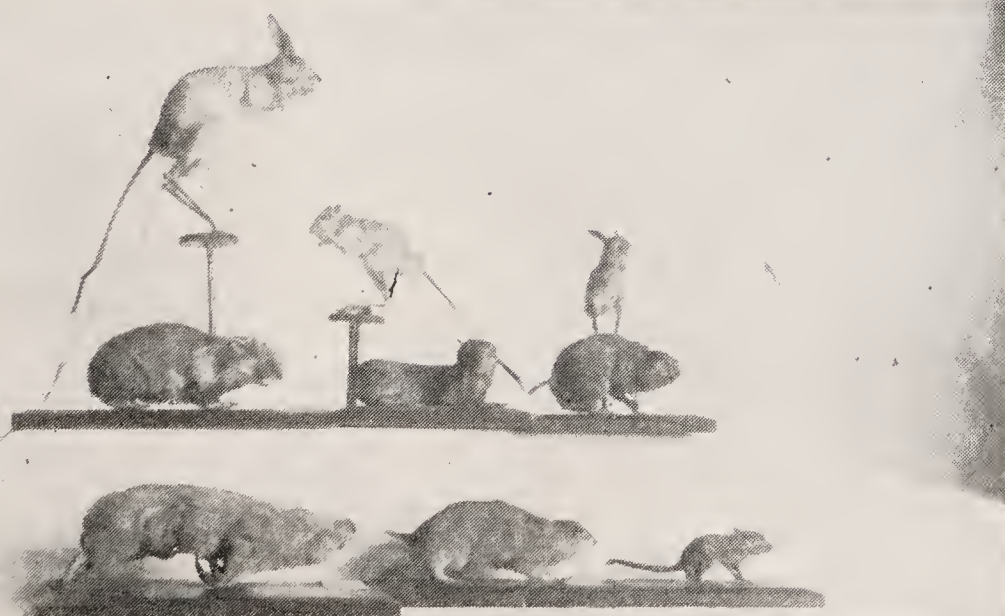
Performing Plague Inhalation Experiments in open air, Harbin 1922.



Hibernating tarabagan, held in the hand, Harbin, February, 1922.



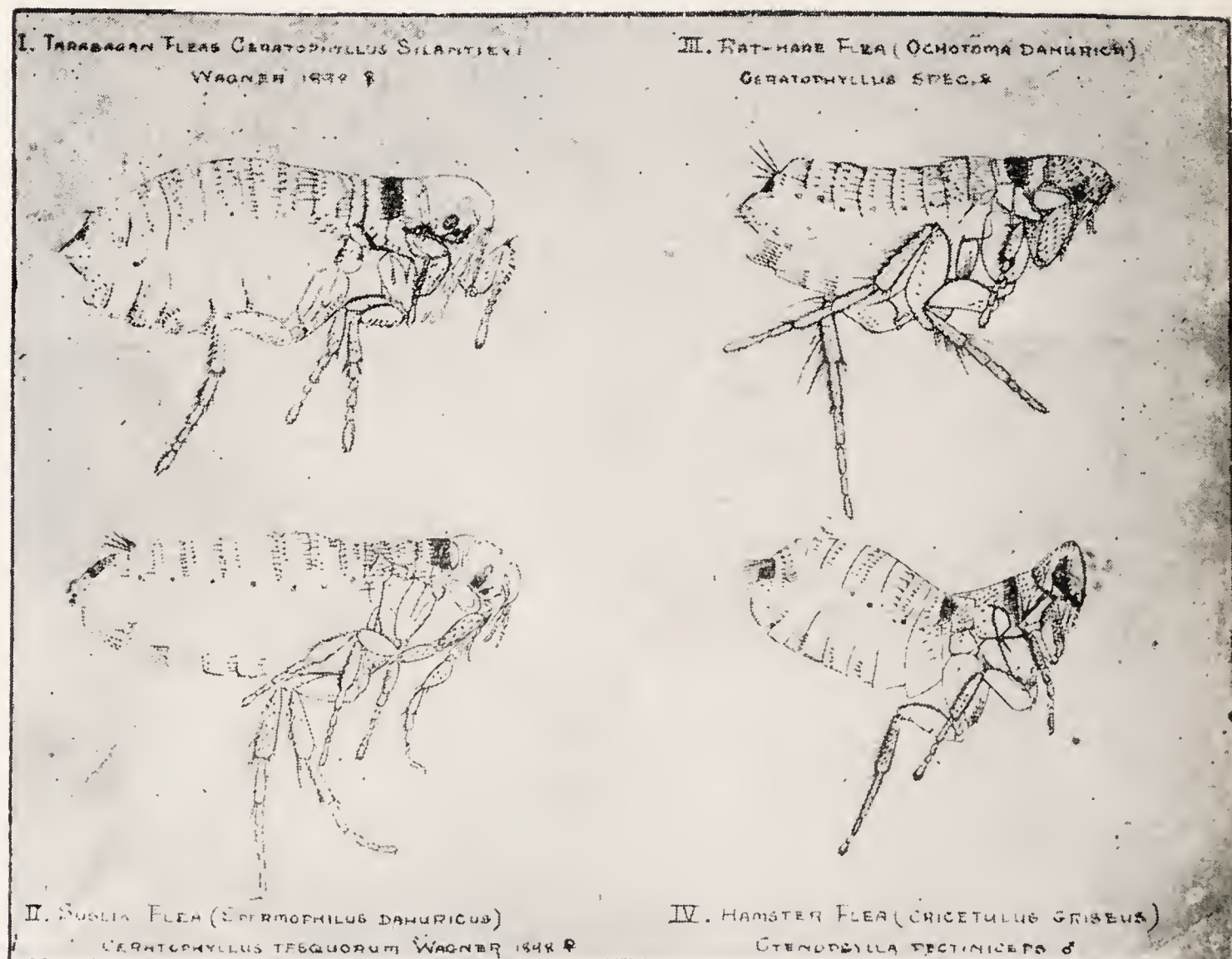
California ground squirrels. (Presented by Dr. J. G. Geiger, Commissioner of Health, San Francisco).



Some wild rodents of Manchuria and U.S.S.R.



A corner of the Harbin Laboratory of the Manchurian Plague Prevention Service.



Fleas of Manchurian wild rodents.



Photograph of a live tarabagan (*Arctomys bobac*). Note the savage appearance and strong claws.



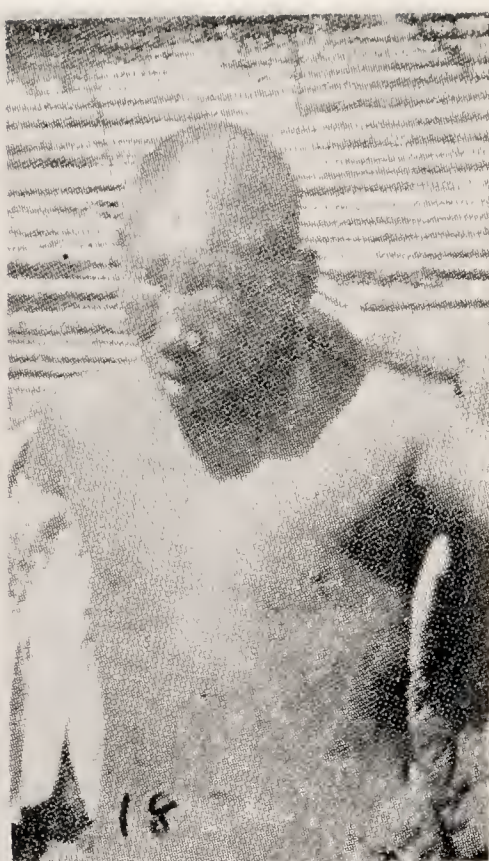
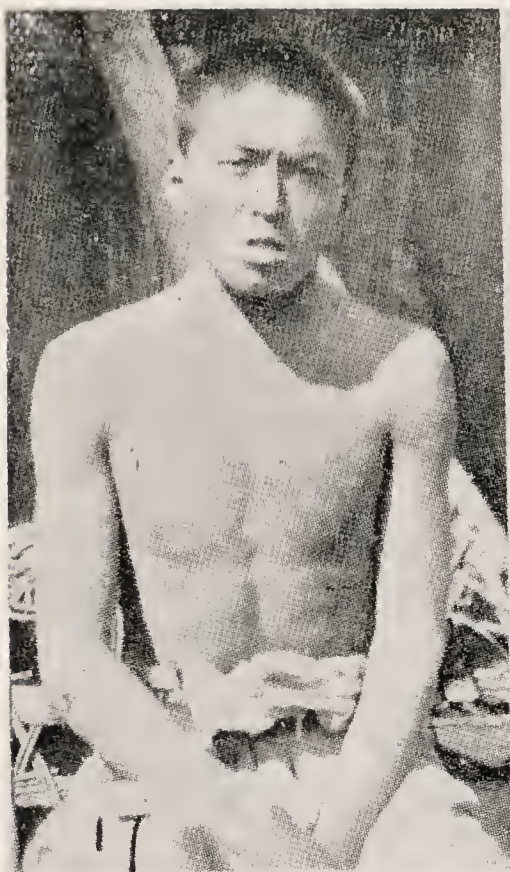
Types of plague-infected house, Fuchiatien, Manchuria, 1911, showing mud-and-straw walls, thatched roofs, paper windows and mud-and-straw sleeping *kangs*.

(The old woman and child were the only survivors out of a family of eight living in the hut on the right, during the Pneumonic Plague Epidemic of 1911).

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Post-mortem of plague bodies in open field, Tungliao epidemic, 1928.



Types of bubonic plague in South Manchuria, 1929.

17. Double cervical. 18. Left axillary.

19. Right femoral. 20. Double inguinal.

